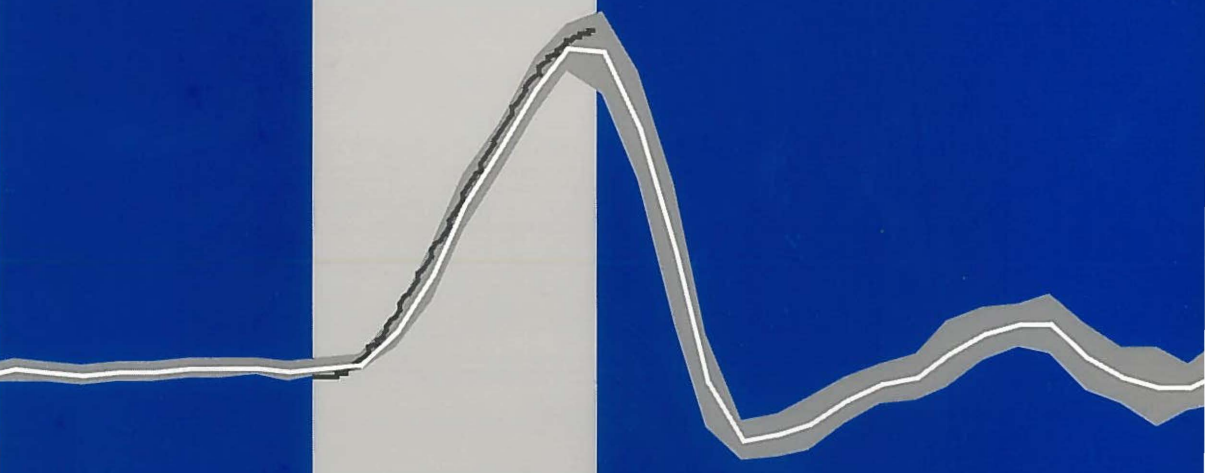
control of unexpected jaw-closing movements



Geranda Slager

CONTROL OF UNEXPECTED JAW-CLOSING MOVEMENTS

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STELLINGEN behorende bij het proefschrift
CONTROL OF UNEXPECTED JAW-CLOSING MOVEMENTS

De kracht-snelheid-eigenschappen van spiervezels zijn in eerste instantie verantwoordelijk voor het opvangen van snelle houdings- en bewegingsverstoringen.

Computermodellen dienen in wetenschappelijk onderzoek gericht te zijn op inzicht en niet op volledigheid.

De aanwijzing 'snij altijd van je af' in de cursus creatief met kurk is een goed advies, omdat bij het plotseling meegeven van het te snijden materiaal de snijkracht nauwelijks afneemt door de geringe verkorting van de arm- en schouderspieren.

In theorieën over bewegingssturing dient meer aandacht besteed te worden aan de fysiologische eigenschappen van spiervezels en pezige elementen.

Gezien het feit dat 1 op de 3 consultaties bij een huisarts betrekking heeft op het houdings- en bewegingsapparaat, is het aanbevelenswaardig tijdens de opleiding geneeskunde veel aandacht te besteden aan dit onderwerp.

Met de rente van 180.000 miljonairs in Nederland is het inkomen van 1,3 miljoen mensen die op het sociaal minimum leven gemakkelijk te verhogen.

Het feit dat anonieme programmeurs bij Microsoft een zogenaamd 'Easter egg' (met al hun namen) hebben verstopt in het veelgebruikte programma Word 6.0 is een voorbeeld van de drang naar erkenning voor werk dat gedaan is.

Gezien de socialistische grondbeginselen van het christendom, zoals vermeld in het Nieuwe Testament, zouden de klein-christelijke partijen moeten horen tot de klein-linkse partijen.

Onder het motto 'vroeg geleerd, oud gedaan' is het opnemen van ontspannings-oefeningen in het basisonderwijs wenselijk.

Wanneer een medisch student zich verspreekt bij de afkorting van het Gilles de la Tourette Syndroom en spreekt van GTST kan dit wijzen op te veel aandacht voor een soapserie en te weinig aandacht voor de leerstof.

Gecorrigeerd voor het sexe-gerelateerde verschil in maximaal aeroob vermogen rijden vrouwen in een (gemengd) wielerpeloton continu op kop.

3 juni 1998, Groningen

Geranda Slager

This work was carried out at the Department of Medical Physiology,
University of Groningen, the Netherlands

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CONTROL OF UNEXPECTED JAW-CLOSING MOVEMENTS

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in het openbaar te verdedigen op
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Geranda Erica Catharina Slager

geboren op 15 november 1964
te Uithuizen

Promotor: Prof. dr. J.D. van Willigen

Referent: Dr. E. Otten

voor mijn overleden oma
Geertruida Hendrika
Wegstapel-van Lindenberg

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van Willigen JD, Otten E, Slager GEC and Broekhuijsen ML
Contribution of the digastric muscles to the control of bite force in man.
Archs oral Biol., 42, 45-56, 1997.

Nagashima T, Slager GEC, Otten E, Broekhuijsen ML and van Willigen JD
Impact velocities of the teeth after a sudden unloading at various initial bite forces, degrees of mouth opening and distances of travel.
J. Dent. Res., 76, 1751-1759, 1997.

Slager GEC, Otten E, van Eijden TMGJ and van Willigen JD
Mathematical model of the human jaw system simulating static biting and movements after unloading.
J. Neurophysiol. 78, 3222-3233, 1997.

Slager GEC, Otten E, Nagashima T and van Willigen JD
The riddle of the large loss in bite force after fast jaw closing movements.
J. Dent. Res. (accepted), 1998.

Slager GEC, Otten E, Nagashima T and van Willigen JD
Human bite force responses after fast and slow jaw-closing movements.
J. Dent. Res. (submitted)

CONTENTS

Introduction

The bony elements of the human jaw system are the maxilla -which is part of the skull-, the mandible -which is connected to the temporal bone of the skull by two joints-, and the hyoid bone -which serves as an insertion for the hyoidal muscles. Six muscle groups are involved in movements around the mouth, but three groups mainly control the lower jaw and the hyoid-tongue complex: the supramandibular muscle group which closes the mouth, and the supra- and the infrahyoidal muscle group which are involved in jaw opening movements.

The muscles composing the above mentioned muscle groups differ in architecture, and muscle fiber content and thus in muscle strength. The jaw-closing muscles are powerful, the hyoidal muscle week; altogether the jaw-closing muscles are able to exert forces up to 600 N (Pruim et al, 1980; Koolstra et al. 1988; Van Eijden et al., 1990 and 1991) at the level of the front teeth, whereas the maximal opening force exerted by all opening muscles is only about 120 N (Sharkey et al., 1984).

Since the effective mass of the mandible is only about 0.20 kg at the level of the lower front teeth, there is a serious risk of damage to the teeth when biting through hard and brittle food, since the forces during the bite can be rather high and the margin of safety is small (jaw separations during biting are in the order of 2 to 3 cm). For example, it can be calculated that, when biting through brittle food at an initial mouth opening of 20 mm with a constant bite force of 100 N, the teeth will collide with a velocity of 4.5 m/s (i.e. 16 km/h) after breaking of the food.

However, these high velocities do not occur in reality, because in unloading experiments -in which, experimentally, the resistance to a forceful static bite is suddenly withdrawn- it is shown that bite forces decrease at a high rate as soon as the mouth starts closing after the unloading, so that the velocity of the mandible is limited strongly (Miles & Wilkinson, 1982).

There are several possibilities explaining this observation.

Firstly, in concurrence with observations on the limb (Angel et al., 1965; Dufossé et al., 1985; Soechting & Lacquaniti, 1988), reflex events possibly reduce the bite force after a sudden unloading. This idea is supported by Hannam et al. (1968) and Lamarre & Lund (1975) who show that after unloading of a static bite there is an inhibition of the jaw-closing muscles and an activation of the jaw-opening muscles. However, in concurrence with Miles & Wilkinson (1982), it will be shown that these reflex events occur too late to explain the rapid drop in force after the unloading.

Secondly, the rapid drop in force after unloading is possibly caused by co-contraction of the jaw-opening and -closing muscles during the bite. This hypothesis is put forward by Miles & Wilkinson (1982) who suggest that the major factor responsible for limiting the jaw closing movement is a weak contraction of the digastric muscles -which are members of the suprahyoidal muscle group- during the static phase before the unloading. When the resistance to the bite is suddenly withdrawn, the jaw closing movement is arrested by the so-called short-range stiffness (due to distortion of cross bridges between myofilaments; Rack & Westbury, 1974) in the co-contracting digastric muscles.

This hypothesis is -indirectly- supported by Miles & Madigan (1983) who describe that the motor programme executed by the digastric muscles is modified according to the subjects expectation of whether or not the jaw closing yields. In a follow up study Van Willigen et al. (1993) show that subjects fall into three categories: I) Subjects who only vary the level of tonic digastric activity as a function of the experimental situation, II) subjects who co-contract the digastric muscles in line with the masseter muscles; and III) subjects who developed anticipatory strategies by changing the contraction pattern of the digastric muscles as a function of the experimental situation. In the last group, subjects based the modulation mainly on immediate past performance.

The mechanical properties of the jaw muscles, thirdly, may also have profound effects on the dynamics of the bite force after the unloading (Otten 1991; Slager et al., 1995). For example, if the sarcomere lengths of the jaw-closing and -opening muscles are *below* their optimal sarcomere length at the start of the unloading, the force of the jaw-closing muscles will *decrease* (due to the force-length properties of their muscle fibers; Gordon et al., 1966) whereas the force of the jaw-opening muscles will *increase* during the closing movement. The force-velocity properties of the jaw muscles may also add to the reduction in force; the jaw-closing muscles can lose a fair amount of their force when they shorten because less cross bridges will attach to actin filaments during the contraction, resulting in a force reduction. Due to their force-velocity properties, the jaw opening muscles can gain force when they are stretched, since cross bridges need to be broken to stretch the muscle and the stiffness in the muscle is quite high.

The aim of this thesis is to look which factors cause the quick decrease in bite force and so the limitation in the velocity of the mandible after a sudden unloading.

For that we carried out unloading experiments of the jaw, in which subjects were asked to bite isometrically on two bars of which the resistance to the bite was suddenly withdrawn. Initial bite forces, initial mouth openings, distances of travel of the lower jaw when closing as well as velocities of jaw closure were varied. To analyze possible effects of these variables on the force profiles after the unloading the bite, the force exerted on the lower of the two bars and the position of that bar was measured during the experiments. We also recorded surface EMG's from the masseter muscle -that is one of the supra-mandibular muscles-, and the digastric muscles to see how the level of (co-)contraction of these muscle influences the shape of the force profiles.

However, because the actual length and the velocity of the muscle fibers during the experiments is unknown (due to compliance of the tendinous sheets), it is hard to judge how reflex events, co-contraction, force-length and force-velocity properties of the muscle fibers contribute to the decline in bite force during jaw movement from experimental results only. To by-pass this problem we formulated a numerical model of the human jaw system (simulating our unloading ex-periments) to judge the relative contribution of the variables under discussion to the reduction in force. The physiological and anatomical parameter values

of this model were taken from literature and from a data basis of a human cadaver study. The other parameter values were found by fitting the model results to data from a set of unloading experiments. Thereafter the model was verified with new experimental data.

Outline of the thesis

As mentioned before, this thesis deals with the large loss in bite force observed after an unexpected unloading of the jaw.

Chapter 1, 2 and 3 deal, mainly, with the quick drop in force during the *dynamic phase after unloading* until the mandible has come to a standstill, whereas chapter 4 and 5 are dedicated to the observation that in the *post-dynamic phase* the *residual bite force* (i.e. the force when the jaw system is in a static condition again after the impact) is remarkably small.

Chapter 1 describes the contribution of the (co-contracting) digastric muscles to the rapid decline in bite force magnitude after unloading of a static bite. Experiments are described and discussed in which subjects were asked to perform two different biting tasks with a sudden unloading, and correlating the degree of co-contraction of the digastrics (as derived from their EMG's) with the *force and the velocity at impact* (as measured just before the jaw-closing movement was arrested after a course of 5 mm), and the residual bite force.

Chapter 2 reports on a study in which the magnitude of the impact velocity of the mandible -when the lower jaw is arrested after an unloading- is determined as function of 4 different initial bite forces, 4 different initial degrees of mouth opening and 3 different distances of travel of the mandible.

In Chapter 3, a mathematical, forward dynamic model of the human jaw system is described, that simulates static bites and the dynamic phase after unloading of this bite. With this model the influence of reflex-events, co-contraction, force-length properties and force-velocity properties of the jaw-opening and -closing muscles on the velocity at impact were explored. Morphometric data from a cadaver study were used in the model to come to anatomical realism. Physiological input values were mainly taken from literature.

As mentioned before, when the mandible is arrested briefly after an unloading, the residual bite force appears to be unusually small although the jaw-closing muscles are still contracting. This phenomenon can not be explained by the force-velocity properties of the jaw muscles alone. The second part of this thesis deals with this problem.

Chapter 4 describes experiments dealing with the contribution of the initial bite force, the initial degree of mouth opening and the distance of jaw travel to the magnitude of the residual bite force. A possible influence of the force-length properties of the jaw-closing muscles on the magnitude of the residual force was analyzed by a model study. Also, possible influences of reflex events of the jaw muscles on the residual force were analyzed. This was done by direct electrical stimulation of both masseter muscles and measuring the electromechanical delay after their excitation was switched off.

Finally, in chapter 5 the influence of the velocity of jaw closure on the force profile

of the closing mandible after an unloading is described. Data are presented on unloading experiments over four different distances of jaw closure. There were two series of experiments. In one series the jaw could move freely after the unloading, in the other the maximal velocity of jaw closure was reduced by changing the resistance to jaw closure.

The thesis ends with some reflections and an English and Dutch summary.

1

Contribution of the digastric muscles to the control of bite force in man

Summary

We investigated the contribution of the (co-contracting) digastric muscles to the rapid decline in bite force magnitude after an unloading of a static bite. This was done by asking participants to perform two different biting tasks with sudden unloading, and correlating the degree of co-contraction of the digastric (as derived from their electromyograms) with the impact force, the impact velocity (as measured after a travel distance of 5 mm), and the residual force when the jaw system was in static conditions again after the impact. Co-contraction of the digastrics was varied by asking participants to perform the biting task while controlling bite force (force-controlled experiments) or jaw position (position-controlled experiments). In half of the experiments, participants co-contracted their digastrics more strongly in the position-controlled experiments than in the force-controlled experiments. However, there was no clear relationship between the level of co-contraction and the magnitude of the impact force, the impact velocity and the residual force. The results imply that co-contraction of the digastric muscles is not sufficient to explain the reduction in bite force and the low impact velocity after an unexpected jaw-closing movement. Two other possible mechanisms that reduce forces in an unloaded jaw system are: 1) force-velocity properties of the activated jaw muscles in conjunction with creep of the aponeurotic sheets of the jaw muscles, resulting in a slow partial recovery of the biting force after impact; 2) force-length properties of jaw-opening muscles of which we did not record activity.

Introduction

When the resistance to a forceful isometric bite is suddenly withdrawn, the jaw-closing movement is arrested before the teeth come together (Hannam et al., 1968). In this condition an unloading reflex (inhibition of jaw-closing and excitation of jaw-opening muscles) is evoked (Hannam et al., 1968; Lamarre & Lund, 1975; Miles & Wilkinson, 1982; Van Willigen et al., 1995; Yoshida & Inoue, 1995). According to Miles & Wilkinson (1982) the inhibition of the jaw-closing muscles and the reflex activation of the jaw-opening muscles occur too late to explain the rapid arrest of the mandibular movement. They conclude that the phenomenon responsible for limiting the jaw-closing movement is a weak co-contraction of the digastric muscles during the phase of isometric contraction of the jaw-closing muscles; when the resistance to closing is suddenly removed, the rapid jaw-closing movement is arrested by the so-called short-range stiffness (Rack & Westbury, 1974) of the co-contracting jaw-opening muscles.

According to Otten (1991) the force-velocity properties of the active muscles (jaw-closing as well as jaw-opening muscles) are mainly responsible for the limitation of the velocity of the lower jaw after the resistance to the bite force disappears. Due to their force-velocity relations, with the sudden acceleration of the lower jaw, the jaw-closing muscles lose most of their force when they shorten and the jaw-opening muscles increase their force because they are stretched.

Van Willigen et al. (1995) report that only at biting forces smaller than 40 N and distances of travel of the mandible greater than 10 mm, co-contraction of the digastrics is sufficient to arrest the movement of the mandible and to prevent the teeth from colliding. They conclude that 'stiffness increase of the jaw system by co-contraction does not help in reducing the initial build up of the closing velocity of the jaw when the resistance between the teeth declines, since the required net bite force is independent of co-contraction. Co-contraction, however, does make the net bite force decrease at a steeper rate when the jaw closes, so that the impact velocity is lower than in a case where co-contraction is not used'. This statement is supported by Slager et al. (1995). In a model analysis they give evidence that the rapid limitation of velocity of the jaw is due to the combined effects of the force-velocity and force-length properties of the contracting and co-contracting muscles.

We have studied the contribution of the amount of co-contraction of the digastric muscles to the bite-force control of the jaw system. Participants were asked to perform a biting task with sudden unloading, and the degree of co-contraction of the digastric muscles (as derived from their EMGs) was correlated with the reduction in bite force and impact velocity (as measured after the jaw-closing movement was terminated after a course of 5 mm). Co-contraction of the digastrics was varied by asking participants to perform the biting task while controlling either bite force or jaw position. If co-contraction of the digastrics assists in the control of bite force, we would expect higher levels of co-contraction in the position-controlled experiments than in the

force-controlled experiments, associated with a stronger reduction in bite force and impact velocity.

Material and Methods

Six dentate subjects participated in the experiments. All were free from signs of muscular or temporomandibular dysfunction. They were fully informed about the experimental conditions and gave their consent.

Throughout the experiments the participants sat in front of a force transducer/unloading device (Fig. 1). The apparatus was adjusted so that it was at the level of the mouth. Bite forces were exerted between the upper and lower front teeth. Behind the apparatus the participant could see the magnitude of the exerted bite force displayed on an oscilloscope screen; on that screen an oblique line was drawn.

The apparatus contained two parallel aluminum bars, 80 mm long, 50 mm wide and 6 mm thick. For comfort the biting areas of the bars were covered with a thin layer of rubber. The upper bar was attached to two vertical plates mounted on a base plate. The lower bar either hinged around an axis (unloading mode) or was locked by inserting an additional pin in a hole provided (force transducer mode). The lower bar was fixed to the hinging axis on which a magnet was mounted so that the angular displacement of the lower bar could be recorded with a magneto-resistive-sensor module (Philips KM11OBH/21). Two pairs of balanced strain gauges were attached on either side of the lower bar 25 mm from the mouth piece. In this way the difference in torque induced by the bite force could be recorded over a fixed distance and the bite force was measured independently of the point of force impact. The initial resistance to closing was achieved by a solenoid fixed to the lower bar. This solenoid could be switched off either by hand or triggered by the output of the lower bar strain gauges at

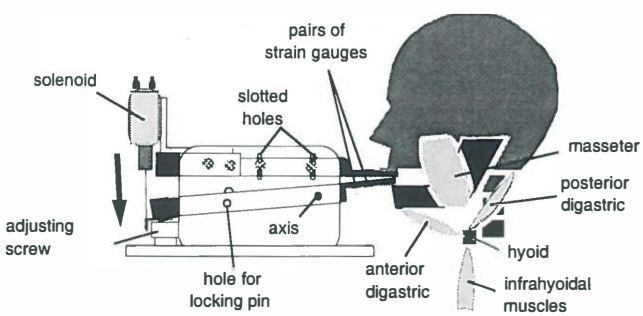


Figure 1. 'Unloading' apparatus and force-transducer combination: two parallel aluminum biting bars (with pairs of strain gauges) are attached to two vertical metal plates mounted on a base plate; the lower bar is fixed to a hinging axis of which the angular displacement is measured; the initial resistance to closing was achieved by a solenoid.

a voltage equivalent to 100, 40 and 10 N. In all experiments the unloading started at an interincisor distance of 19 mm. When the solenoid was switched off, the lower bar dropped at the back, so that the front was lifted up. The distance of travel of the lower bar could be adjusted by means of a screw which was in the base plate at the level of the back of the bar (Fig. 1). By adjusting the screw, care was taken that the interincisor end distance was 14 mm so that the distance of travel of the lower bar was 5 mm in all experiments. We took this course of 5 mm to ensure that the jaw-closing movement was terminated in time, and a residual bite force could be measured. To buffer the shock of collision, the head of the adjusting screw was covered with a soft plastic cap. The output of the pairs of strain gauges was fed into a differential amplifier. The output of the lower bar force transducer could be displayed as a dot on the monitor oscilloscope mentioned. The lower bar force transducer was linear in the range of 5-200 N.

Surface EMGs were recorded from the right masseter muscle and the right anterior digastric muscle using pairs of 10 mm disc electrodes at an interelectrode distance of approx. 10 mm for the masseter muscle, and 10-25 mm behind the mandibular insertion of the anterior digastric muscle. Both pairs of electrodes were connected to the same reference electrode which was placed on the neck. EMGs were amplified (bandwidth DC to 4 kHz) according to the experimental condition [10^4 times (masseter in the 100 N experiments) to 5 x 10^5 times (digastrics in the 10 N experiments), see below]. EMG signals and the output of the position and force transducers were digitized in real time (sample frequency 1 kHz) and stored on hard disk. [With this electrode configuration it unlikely that with either pair of electrodes activity of other muscles than the masseter and the digastric muscle was received, as only muscle fibers of motor units located within about 10 - 12 mm from the electrodes contribute significantly to the surface EMG (Fuglevand et al. 1992).]

EMG values of maximal voluntary contraction (MVC) of the masseter muscle were obtained by encouraging the participants - with unchanged electrode arrangement- to bite three times isometrically as hard as possible on the locked apparatus for 5 seconds. Between the recordings there was a 1 min. pause. For the digastrics the mandible was stabilised by hand and the participants were asked to contract their jaw-opening muscles as hard as possible also three times for 5 s. The recorded EMGs were rectified and averaged (running average, window 1 s); the highest average value found was called 100% MVC; EMGs were normalised relative to this value.

There were two groups of experiments: (1) force-controlled and (2) position-controlled experiments. (1) In the force-controlled experiments the participants were asked to perform a biting task while controlling their bite force. This was achieved by asking them to track the ramp as drawn on the oscilloscope screen by biting on the two bars. In this case the dot displaying the output of the lower force transducer moved with a velocity of 10 mm/s; the slope of the oblique part of the track was such that a biting trial took 6.0 s. The experimental conditions were: biting the ramp with a

sudden unloading at 100, 40 or 10 N. The start of the task was signalled by the appearance of the force output dot on the oscilloscope screen. (2) In the position-controlled experiments jaw position was the controlled variable. Here the participants were asked to bite without visual guidance (eyes closed) through a resistance of 100, 40 or 10 N with care; they were encouraged to co-contract their digastrics voluntarily and asked to hold the mandible at its position after the resistance to the bite force was suddenly removed.

Data analysis

The normalised EMGs were rectified. As a measure of co-contraction of the digastric muscles we took the average of these rectified digastric EMGs over a period of 50 ms prior to the moment the solenoid was switched off.

To establish delays of silent periods and reflexes we looked in the EMG signals for a period of time greater than 20 ms in which each sample of the signal fell below (or exceeded) a threshold set at 20% of the maximum of the EMG at any time in that recording. Delays relative to the start of unloading could then be established as the time elapsed after unloading until the start of the silent period. Figure 2A gives examples -in a time domain 200 ms before and 300 ms after unloading in a 100 N force-controlled experiment- of the rectified EMG of the masseter and of the digastric muscle (upper panels), and recordings of the bite force and the position of the lower bar (lower panels). Note that the (time) averaged EMGs (thin lines in the EMG displays) are 60% MVC for the masseter muscle and 3% MVC for the digastric muscle, although peak values in the EMGs are well above 100% MVC.

As critical measures of the ability of a subject to handle an unexpected unloading of the lower jaw, we examined (1) the impact force as measured by the lower bar force transducer when the lower bar hit the plastic cap of the adjusting screw, (2) the impact velocity of the lower bar and (3) the residual force after the impact when the jaw/bar system was in static conditions again. Values for these parameter were obtained per subject from averages (8-20 trials) of each of the 6 experimental conditions.

Figure 2B shows a magnification of the force (top panel) and position recordings (bottom panel) of Figure 2A and the (calculated) velocity of the lower bar (middle panel). Point 1 in the top panel indicates the moment of unloading and the magnitude of the bite force at unloading. We established the value of the impact force by looking for the first minimum in the force signal after the solenoid was switched off (point 2 in the force recording of Fig. 2B). As a non-zero force recording implies that the teeth are in contact with the lower bar, the velocity of the teeth is the same as that of the lower bar. Impact velocities (point 5 in the velocity display of Fig. 2B) were established by differentiating the position signal of the lower bar, smoothing the result with a moving window of 5 ms and taking the maximum value during bar travel. We established the value of the residual forces (point 4 in the top panel of Fig. 2B) using the following algorithm. The average force curve (thin curve in the top panel of Fig. 2B) of each subject in a particular experimental condi-

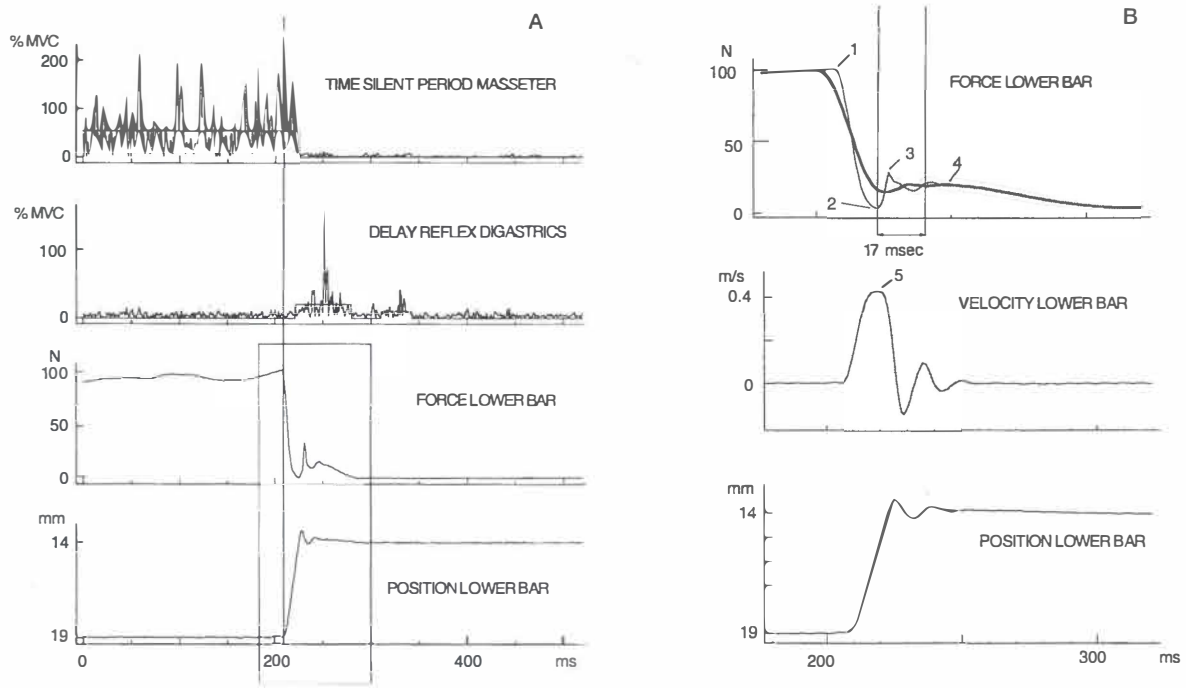


Figure 2. Illustration of the outcome of an analysis, as described in Material and Methods, of a 100 N force-controlled experiment. (A) Upper panels: a trace of the rectified raw masseter EMG (relative to the averaged maximal voluntary contractions) and the onset of the silent period. Below is a trace of the rectified raw digastric EMG (relative to the maximal voluntary contractions) and the onset of the digastric reflex. Lower panels: force signal and the position signal of the lower bar. (B) Magnification of the force and position signals identified by the boxed area of (A). The panels show: the average with SD (grey areas) of the lower-bar bite force signal (average, thin line; filtered, thick line), and the velocity and the position of the lower bar. Top panel: (1) time of unloading and bite force at unloading; (2) time of impact and magnitude of impact force; (3) transient force peak due to collision of lower bar to head of adjusting screw, and (4) time of occurrence of the maximal residual force and its magnitude. Middle panel: (5) magnitude of impact velocity.

tion was low-pass filtered with a running window of 16 ms (thick curve in the top panel of Fig. 2B). We then looked for the first maximum in this averaged force curve at least 17 ms after the moment of impact-; this value was taken as a measure of the residual force. The boundary of 17 ms was taken in order to exclude transient force peaks caused by the collision of the back of the lower bar with the head of the adjusting screw shortly after impact (point 3 in the top panel of Fig. 2B).

Pearson's correlation was used to detect possible differences between the various experiments. All processing and graphics were performed by an Apple Macintosh IIcx computer system running 'DataMonster 2.0' a data analysis, graphics and simulation program written by one of us (E.O.).

Results

The experiments

In all the experimental conditions all participants co-contracted their digastric muscles during the bite above their baseline activity (Figs 3 and 4). In general the participants showed the following behaviour: they opened the mouth and made contact with the upper and lower bar of the unloading device (Figs 3 and 4, section B) and co-contracted their digastrics in anticipation of the task to be performed (Figs 3 and 4, section C); they then increased their biting force and performed their task (Figs 3 and 4, section D); depending on the experimental situation, the level of co-contraction of the digastrics was between 2 and 24% MVC.

With the force-controlled experiments participants had difficulty in tracking the ramp smoothly as can be seen from the variations in the bite force track relative to the ramp to be tracked (Fig. 3). After unloading, the force dropped almost to zero and recovered only partially when the bar reached a new equilibrium in its end-position. In all experiments with initial bite forces of 40 and 100 N there was a silent period in the masseter EMG after the jaw started to move. Sometimes some increase in activity of the digastric muscle could be observed after impact.

With the position-controlled experiments, participants increased their biting force slowly and explored the resistance to closing by periodically varying the activity of their jaw-closing muscles (Fig. 4, which are recordings of the same individual as in Fig. 3); the digastric muscle co-contracted during the whole bite in synchrony with the masseter muscle (Fig. 4, section D1 and D2).

When comparing Figs 3 and 4, a high similarity can be observed in the dynamic phase of the experiment, although the muscular events in the static phase were quite different.

Reflex events

A silent period in the masseter EMGs could be observed 9-20 ms after unloading in all 100 N and 40 N experiments; thereafter sometimes a second burst (delay *ca* 75 ms; duration *ca* 150 ms) was seen. However, with the 10 N trails only two participants occasionally displayed a silent period (latency about 20 ms) in the masseter signal. In almost all experiments an increase in digastric activity could be observed with a

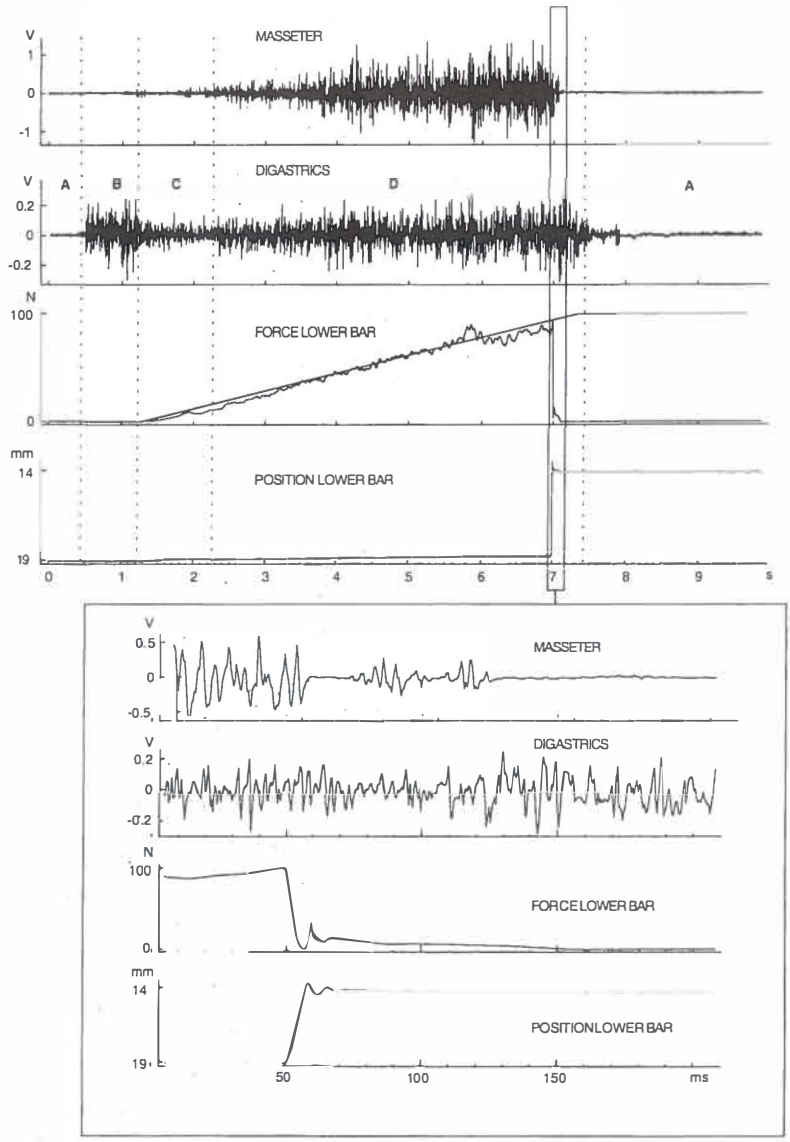


Figure 3. Traces showing EMGs of the right masseter and anterior digastric (amplified 10^3 and 10^4 times, respectively) and recordings of the bite force at the lower bar of a participant tracking a ramp on an oscilloscope screen by biting on the bars of the device shown in Fig. 1. At 100 N a sudden unloading of the jaw occurred. Initial incisor separation: 19 mm; end position: 14 mm. The bottom panel gives the events around the moment of unloading at a smaller time scale. A silent period in the masseter EMG can be seen at *ca* 10 ms after the unloading; there is also some increase in the digastric EMG after 60 ms.

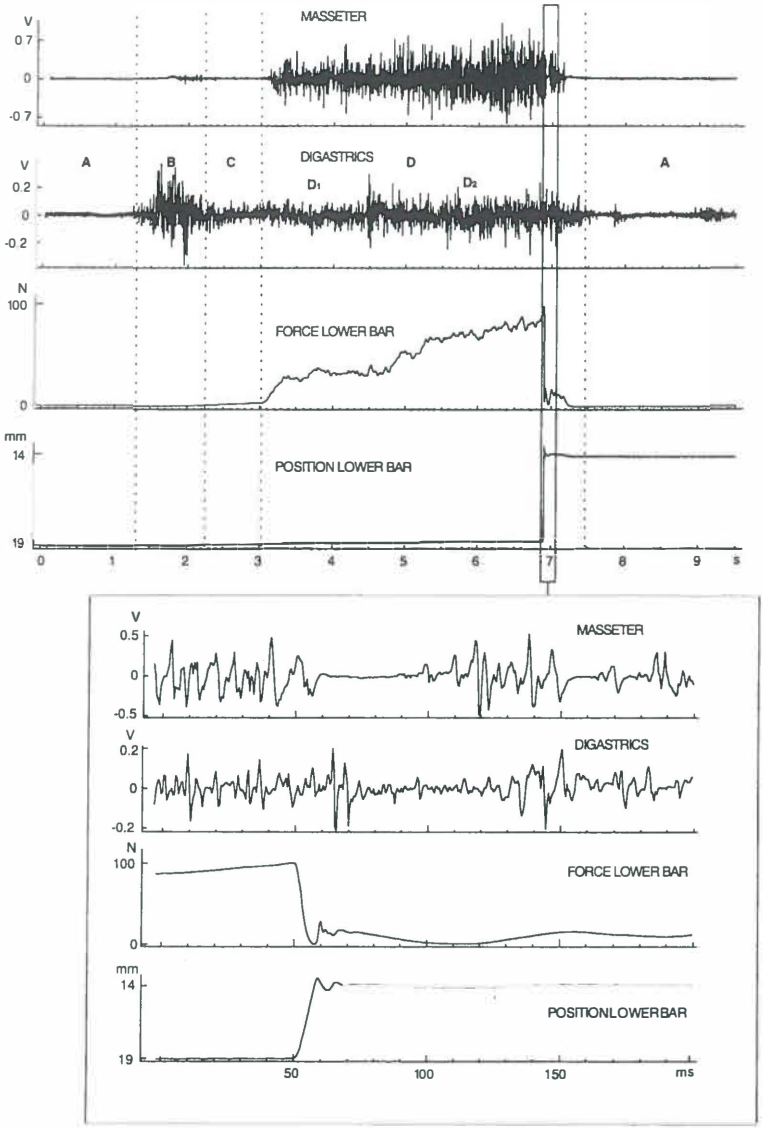


Figure 4. Traces showing EMGs of the right masseter and anterior digastric (amplified 10^3 and 10^4 times, respectively) and recordings of the force of the lower bar of same participant as Fig. 3, carefully biting through a resistance of 100 N with the eyes closed. Initial incisor separation: 19 mm; end position: 14 mm. The participants were encouraged to co-contract their digastrics voluntarily and asked to hold the mandible at its position after unloading. Note that they increased their biting force slowly and explored the resistance to closing regularly by varying the activity of the jaw-closing muscle; the digastric co-contracted during the whole bite in synchrony with the jaw-closing muscles. The panel at the bottom gives the events around the moment of unloading at a smaller time scale. A silent period in the masseter and digastric EMG can be seen at *ca* 12 ms and 25 ms after the unloading; thereafter the digastric increases its activity.

variable latency (5-80 ms) and variable duration (10 to over 450 ms). In general latencies became longer with decreasing initial bite forces in both digastrics and masseters. Reflex events in the force-controlled conditions were comparable to those in the position-controlled circumstances. Table 1 shows the averaged latencies for the masseter silent periods and the digastric reflexes for all participants and all experimental situations.

Relationship between the co-contraction of the digastrics, the impact force and impact velocity, and the residual force

Figure 5 gives an overview of the average co-contraction of the digastrics, the impact force, the impact velocity and the residual force in both experimental conditions. The oblique line is the line of equality in both conditions.

*Co-contraction (Fig. 5*A). As compared to the force-controlled experiments, in the position-controlled experiments half of our participants co-contracted their digastrics stronger: about 150% stronger in the 40 and 10 N experiments and about 20% stronger in the 100 N experiments.

*Impact force (Fig. 5*B). Impact forces were between 9 N (for the 100 N experiments) and 0 N (for the 10 N experiments). In 5 of the 18 pairs of experiments the impact force in the force-controlled experiments was slightly higher (up to 4 N) than that in the position-controlled experiments. In the other cases impact forces were not significantly different in the two conditions.

Impact velocity (Fig. 5C). There were no significant differences detectable between the impact velocities in the two ex-

Table 1. Delays of silent periods in the masseter and of digastric reflexes evoked by a sudden release in bite force (± SD) for all experimental conditions.

	Participants No.					
EXPERIMENT	1	2	3	4	5	6
Delay silent period masseter (ms)						
FC 100 N	9±6	11±5	16±6	14±5	15±5	10±2
FC 40 N	18±7	17±3	15±8	17±7	14±13	13±8
FC 10 N	–	17±6	–	–	23±8	–
PC 100 N	12±8	13±3	10±3	15±8	15±4	12±4
PC 40 N	20±8	12±11	13±6	17±9	16±11	14±8
PC 10 N	–	–	–	–	24±5	–
Delay digastric reflexes (ms)						
FC 100 N	17±10	36±8	9±2	28±3	13±5	20±7
FC 40 N	7±4	50±26	12±4	25±10	–	45±30
FC 10 N	59±18	48±12	45±31	49±21	–	48±13
PC 100 N	5±7	37±11	14±6	24±1	15±6	79±16
PC 40 N	10±4	32±13	13±6	16±3	29±15	72±23
PC 10 N	54±27	29±10	56±12	82±10	–	75±44

FC, force-controlled experiments; PC, position-controlled experiments.

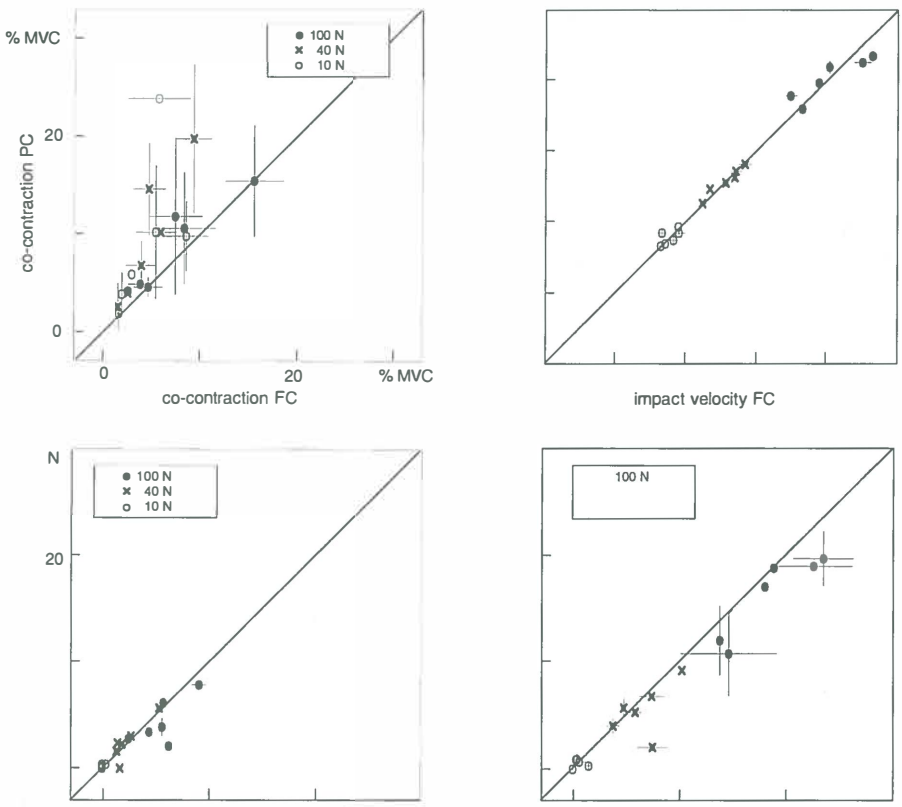


Figure 5. Overview for all experimental conditions. (A) Average co-contraction of the digastric muscles (as percentages of maximal voluntary contraction) in the position-controlled condition (and its SD) plotted against the average co-contraction observed in the force-controlled condition. Note the higher levels of co-contraction in half of the position-controlled experiments. (B) Average force at impact in the position-controlled condition (and its SD) plotted against that observed in the force-controlled condition. Note that in some cases the impact force is lower in the position-controlled experiments. (C) Average velocity at impact of the lower jaw in the position-controlled condition (and its SD) plotted against that observed in the force-controlled condition. Note that there is no consistent difference between the two conditions. (D) Average maximal residual force after the dynamic phase (and its SD) in the position-controlled condition plotted against that observed in the force-controlled condition. Note that in some cases the residual force is lower in the position-controlled experiments. The oblique line is the line of equality in both conditions. •, 100 N experiments; **x**, 40 N experiments; **0**, 10 N experiments. Fimpact, impact force; Fresidual, residual force; FC, force-controlled experiments; PC, position-controlled experiments.

perimental conditions; the largest differences being about 7% in either direction. Obviously there is no unique relation between impact force and impact velocity.

Residual force (Fig. 5D). In 5 of the 18 cases the residual forces were higher in the force-controlled experiments as compared to the position-controlled ones. In the other cases, there was no significant difference between residual forces measured in the two conditions.

Effectiveness of co-contraction on handling of unloading

Figure 6 shows three panels, in which the differences in impact force, impact velocity and residual force between the two sets of experiments are plotted as functions of the differences in co-contraction. The differences were calculated by subtracting the values of the force-controlled experiments from those of the position-controlled experiments. The panels illustrate the effectiveness of an increase in co-contraction on the handling of the sudden unloading. As can be seen, a substantial increase in co-contraction is hardly ever accompanied by decreases in the impact force, impact velocity or residual force; no particular pattern is noticeable, which is illustrated by the low Pearson's correlation of rP = 0.12, rP = 0.07 and rP = 0.37, respectively.

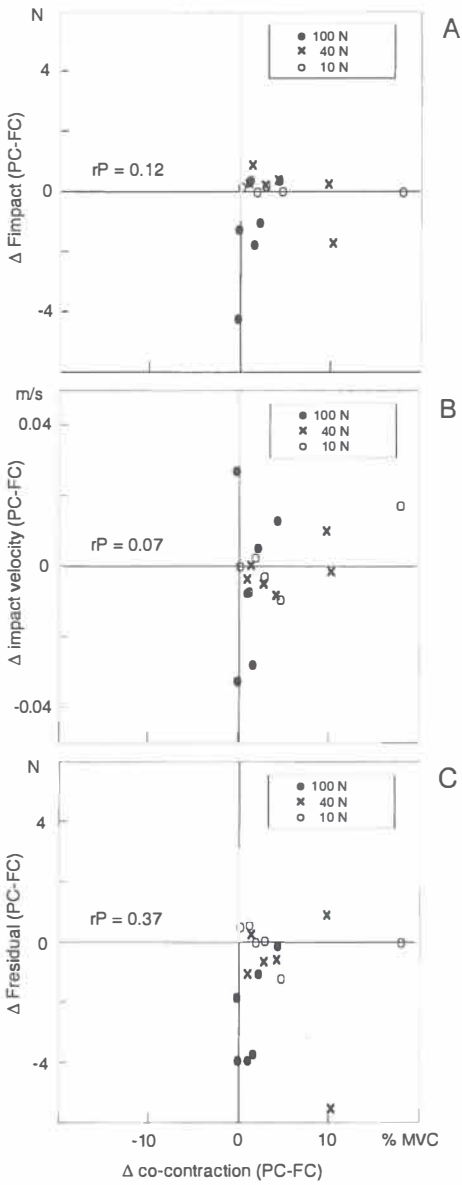


Figure 6. Plots for all participants of the differences in impact force, impact velocity and residual force between the position-controlled and force-controlled experiments as functions of the differences in co-contraction of the digastric muscles in those conditions. Note that none of the variables is systematically effected by differences in co-contraction. rP, Pearson's correlation coefficient. •, 100 N experiments; x, 40 N experiments; o, 10 N experiments. Fimpact, impact force; Fresidual, residual force; FC, force-controlled experiments; PC, position-controlled experiments.

Discussion

In line with Miles & Madigan (1983) and our previous observations (Van Willigen et al., 1993) we observed that participants always co-contracted their digastric muscles weakly (between 2 and 24% MVC) when they expected that the resistance between the teeth would disappear. Similar patterns of co-contraction are described for the interosseous muscles in unloading experiments with the index finger by Kalunzy & Wiesendanger (1992). In concurrence with the observations of Buchanan & Lloyd (1995) on forearm muscles, in position-controlled tasks we observed higher levels of co-contraction of the antagonist (in our case digastric) muscles than in force-controlled tasks. Comparable task related effects are also described by McMillan & Hannam (1992) for the masseter motor unit reflex.

In order to consider motor control as possible candidate for the reduction in bite force and impact velocity, let us consider the reflex events first. The values of the latencies of the digastric reflex burst and the onset of the silent period in the masseter muscle after the unloading are comparable with those measured by Hannam, Matthews & Yemm (1968), Goodwin & Luschei (1974), Lamarre & Lund (1975), Miles & Wilkinson (1982), Van Willigen et al. (1995) and Yoshida & Inoue (1995). In line with Lamarre & Lund (1975) we sometimes observed a polyphasic depression in the EMG-activity of the masseter. In almost all 10 N cases there was no silent period in the masseter, whereas the delay of the digastric reflex was increased markedly. We found no differences in latencies of the digastric reflexes under force-controlled and position-controlled conditions, which is in agreement with Dufosse et al. (1985).

According to Bellemare et al. (1983), in the human slow-fiber soleus muscle, muscle force reaches its 50% level about 100 ms after it becomes electrically silent. We measured silent periods with latencies of at least 9 ms in the masseter, and maximal residual forces (in the 100 N experiments) at about 35 ms after unloading. At this time the masseter may have lost only a fraction of its force due to the onset of the silent period, since only about 25 ms have elapsed of the 100 ms needed to lose 50%. The silent period, being part of the motor control of the lower jaw, is therefore incapable of inducing any significant loss in bite force. The same holds for the impact forces of the lower jaw, since the sum of reflex latencies and electro-mechanical delay of the jaw muscles puts a controlled loss in muscle force at impact well after the observed immediate low level in bite force observed.

According to Miles & Wilkinson (1982) co-contraction of the digastric during a forceful bite provides the jaw system with a mechanism for dealing with unexpected events. When the mandible is suddenly accelerated the active digastric muscles are stretched and increase their force due the resistance of the cross-bridges fastened to their myofilaments (short-range stiffness; Rack & Westbury, 1974). Miles & Wilkinson (1982) state that '.... so it is probably feasible that even a small increase in digastric activity could have increased its stiffness sufficiently to oppose the initial high velocity closing movement'. However,

the effects of short-range stiffness disappear at increases in length of more than 0.25 mm of the digastrics, since the operating range of cross bridges is only about 12 nm per half sarcomere (about 1%) for lengthening (Flitney & Hirst, 1978), and the fiber length of the digastric muscles is about 25 mm (T.M.G.J. Van Eijden, personal communication). In our experiments, the front teeth move 5 mm, which gives a lengthening in digastric fibers of about 2.5 mm, which is 10 times the operating range of the cross bridges. Consequently, short-range stiffness can not be the predominant factor in explaining force reduction.

The average residual force in the 100 N experiments is 18 N; so 82 N is lost over a distance of 5 mm. This loss may be due to a combination of the force-length properties of both the opening and the closing muscles. However, we performed experiments at an initial mouth opening of 19 mm, so that -since the maximal closing force in humans is reached at about 17 mm and hardly varies between 14 and 20 mm (Mackenna & Türker, 1983)- only a small decrease in isometric biting force can be expected from the closing muscles due to their force-length properties.

One can argue that -although hardly comparable to the human masticatory system- in the opossum (Thexton & Hiiemae, 1975) and in the cat (MacKenna & Türker, 1978) the active force generation of the jaw-closing muscles diminishes with smaller gapes. However, the opossum and cat have a shearing and cutting way of feeding in contrast to grinding in man. This is reflected in the force-length curves of the jaw-closing muscles of the opossum and cat, which are more favorable to reducing bite force in sudden closures.

Sharkey et al. (1984) measured, for all human jaw-opening muscles together, an average maximal opening forces of 118 N. We measured co-contraction levels at a maximum of 24% of the MVC in the digastrics. If we assume that all opening muscles contract at this 24% level, the force generated by all opening muscles in our experiments will be maximally 28 N. If the opening muscles were responsible for generating the measured loss in force, they would have to increase their force by a factor of 4, while being stretched by only 10% (see above). This is unheard of in muscle physiology.

Given the above exclusions of (1) motor control, (2) short-range stiffness and (3) force-length properties of muscles as factors in being largely responsible for the observed loss in force after sudden jaw closure, we would like to put forward some alternative possibilities.

Firstly, our earlier suggestion of the effect of force-velocity properties of the jaw muscles on the loss in force may still be useful. One may argue that after the movement, the force-velocity properties can not be operative, as no velocity remains. But in combination with the fact that human jaw-closing muscles have long aponeurotic sheets, (T.M.G.J. Van Eijden, personal communication), we know that the end of the movement of the jaw does not coincide with the end of the movement of the muscle fibers; they may still have to reach their final shortened state, pulling in series with the creeping tendinous sheet. In that case, there is still some loss in force, because the

fibers keep on shortening.

Secondly, other opening muscles from which we did not pick up signals, may be active during the bite and, in their static force-length properties, contribute to the observed force reduction. This requires strong opening muscles with steep force-length properties. A possible candidate, judged by its architecture and physiological cross-section, is the mylohyoid. However, as discussed above, this factor can only be partially responsible for the observed loss in force, due to extreme requirements for the force-length curve.

In summary, we have shown that:

1) Half of the participants studied co-contract their digastric muscles more strongly in a position-controlled than in a force-controlled task under otherwise equal circumstances.
2) There is no clear relationship between the level of co-contraction of the digastrics and the ability of the participants to handle unexpected unloading of their jaws, by reducing impact force, impact velocity and the residual force.
3) The timing of the reflexes in the digastric and the depression of the activity of the masseter were not task-dependent. The latency of these reflex events was highly variable, and in any case too long to account for the loss in force and impact velocity observed.
4) By excluding force-length properties, short-range stiffness and motor control of the opening and closing muscles, we suggest that a combination of force-velocity properties and intramuscular creep may account for the observed loss in force with a possible minor role for opening muscles deeper in the hyoid-mandible complex.

Acknowledgements

The authors are grateful to Mr. J. Mast and Mr. K. Vaartjes for designing and building the unloading apparatus and its control units, and for their help during the experiments.

2

Impact velocities of the teeth after a sudden unloading at various initial bite forces, degrees of mouth opening and distances of travel

Summary

A potential dangerous situation arises when biting on hard and brittle food which suddenly breaks, since the impact velocity of the lower teeth onto the upper teeth after breaking of the food can be high and may cause dental damage. The present experiments were designed to study the magnitude of the impact velocity after a sudden unloading at various initial bite forces, degrees of mouth opening and distances of travel. Subjects were asked to perform a static biting task during which the resistance to the bite was suddenly removed. The upward mandible movement was arrested after a certain distance. The velocity of the lower teeth at impact was calculated just before the mandible came to standstill in combination of 4 different bite forces (100, 80, 60 and 40 N), 4 different initial degrees of mouth opening (33.5, 30.5, 27.5 and 24.5 mm) and 3 different distances of travel of the mandible (4.5, 3.0 and 1.5 mm). We found that the bite force rapidly declined after the unloading, resulting in a small impact velocity of the lower front teeth. This impact velocity largely depended on the magnitude of the initial bite force and the distance traveled; it was hardly sensitive to variations in degree of initial mouth opening. The maximal velocity of the lower teeth was 0.43 m/s (at an initial bite force of 100 N). This maximum was reached after a distance of travel of about 4 mm in 12 ms. The data suggest that the rapid decline in bite force coupled with a limitation of impact velocity is due to the force-velocity properties of the active jaw muscles and is not caused by neural control.

Introduction

In theory, high bite forces cause high impact velocities of the lower teeth onto the upper teeth when biting through hard and brittle food. However, in reality, high bite forces decrease quickly as soon as the mandible starts to move, as has been shown in unloading experiments in which the resistance to a forceful static bite is suddenly withdrawn (Hannam et al., 1968; Miles & Wilkinson, 1982; Van Willigen et al., 1997). Consequently, an almost constant, small velocity of the mandible is reached (Slager et al., 1995), since jaw acceleration returns to zero when net muscle force has vanished and with it the measured bite force. The quick reduction in bite force and the limitation in jaw velocity can not be explained by a sudden inactivity of the jaw-closing muscles (delay of 5-20 ms) or an increased activity of the jaw-opening muscles (delay of 20-40 ms) after the mouth starts closing (Hannam et al., 1968; Lamarre & Lund, 1975; Miles & Wilkinson, 1982; Yoshida & Inoue, 1995; Van Willigen et al., 1997), since reflexes were recorded well after the bite force had vanished.

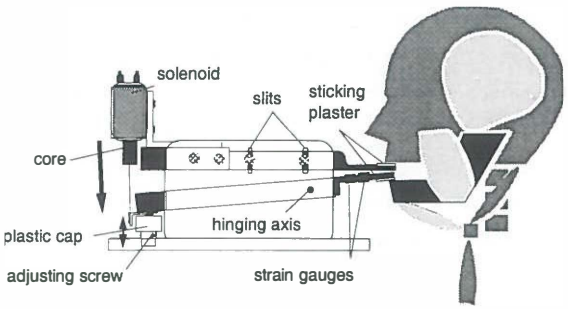
There are several hypotheses explaining the sudden decrease in bite force and the resulting limitation in jaw velocity. From unloading experiments, it has been shown that the jaw-opening (digastric) and jaw-closing (masseter) muscles co-contract during the static phase of the initial bite (Miles & Wilkinson, 1982). These authors suggest that the resistance to elongation of the activated digastric muscles is responsible for the limitation in jaw closing. This resistance is perhaps due to distortion of cross-bridges between myofilaments (Rack & Westbury, 1974).

Otten (1991) stresses the role of the force-velocity properties of the active muscles in the limitation of the velocity of the lower teeth: the jaw-closing muscles lose a fair amount of their force when they shorten, and the opening muscles increase their force when they are stretched, resulting in vanishing of the bite force.

This statement is supported by Slager et al. (1995) who showed that the limitation of velocity of the teeth can be attributed, both, to the force-length and the force-velocity characteristics of the jaw-closing and -opening muscle fibers.

Van Willigen et al. (1997) found in experiments comparable to those of Miles & Wilkinson (1982) that there is no statistic relation between the drop in bite force and the amount of co-contraction of jaw-opening and -closing muscles.

The present experiments were designed to obtain a better insight into the relationship among (1) the initial bite force, (2) the degree of mouth opening and (3) the amount of jaw movement, and the velocity of the lower teeth at impact. We asked subjects to perform a static biting task on a pair of rigid bars, the lower of which could be suddenly released so that an unexpected jaw-closing movement was evoked. The movement of the lower bar and forces exerted on it were recorded. The impact velocity of the lower teeth/lower bar combination was determined under all combinations of four initial bite forces, four initial degrees of mouth opening and three different distances of travel.



unloading apparatus

Figure 1A. Apparatus used as "unloading" apparatus: Two parallel aluminum bars (the lower with pairs of strain gauges) are attached to metal plates mounted on a heavy support; the lower bar is fixed to the hinging axis, the angular rotation of which was measured; the initial resistance to closing was achieved by means of a solenoid.

Material and methods

Participants

Five dentate subjects participated in the experiments. All subjects had no signs of muscular or craniomandibular dysfunction. The subjects were fully informed about the experimental conditions. They gave their informed consent and participated in a protocol that complied with Dutch law.

Throughout the experiments, the subjects sat in front of an unloading device as shown in Figure 1A. The position of the unloading device was adjusted so that it was at the level of the subject's mouth. Bite forces were exerted between the upper and lower incisors and cuspids.

Unloading apparatus

The apparatus contained two parallel aluminum bars (80 mm long, 50 mm wide and 6 mm thick), the upper of which was attached to two vertical plates mounted on a ground plate. The lower bar hinged around an axis. The angular rotation of the lower bar was recorded with a magneto-resistive-sensor module (Philips KM11OBH/21), fixed to the axis of the bar. From the angle of rotation, the displacement of the bitten end of the bar could be calculated.

Two pairs of balanced strain gauges were attached to the upper and lower side of the lower bar at 25 mm from the mouth piece, measuring the bite force applied to the lower bar (Stegenga, 1991). For each subject's comfort, the biting area of the bars were covered with a layer of sticking plaster.

The initial resistance to closing was achieved by an empowered solenoid, the core of which was fixed to the lower bar by means of a steel cable; the solenoid itself was fixed to the body of the device. The movement of the lower bar was prevented by the core of the solenoid, being held in place by the magnetic field of the solenoid.

The magnetic field of the solenoid could be switched off -triggered by the output of the lower bar strain gauges- at a voltage equivalent to 100, 80, 60 and 40 N or by hand.

The position of the upper bar could be varied between experiments by means of slits and bolts so that the initial mouth opening of the subjects at the start of an experiment could be adjusted between 24.5 and 33.5 mm when they bit on the device. The mouth opening of the subjects was defined as the interincisor distance added to the vertical overlap of the dentition (overbite).

After the solenoid was switched off, the (bite) force on the bitten end of the lower bar was suddenly not balanced by a static counter force, and found a dynamic counter force produced by the acceleration of the bar; as a result, the lower bar dropped at the back, and its front was elevated.

The distance traveled of the bitten end of the lower bar could be varied by means of an adjusting screw which was mounted in the ground plate at the level of the end of the lower bar (Fig. 1A). As a buffer for the shock of collision, the head of the screw was covered with a soft plastic cap. [We took small distances of travel because a previous study (van Willigen et al., 1997) showed that at initial bite forces of 10-100 N the force disappears for 90-100% after a distance of travel of 5 mm, resulting in a constant velocity.]

Protocol

The subjects were asked to bite without visual guidance (eyes closed) through a resistance of 100, 80, 60 or 40 N with care; this was done with an initial mouth opening of 24.5, 27.5, 30.5 or 33.5 mm and a distance of travel (after unloading) of 4.5, 3.0 or 1.5 mm. This gave 48 experimental

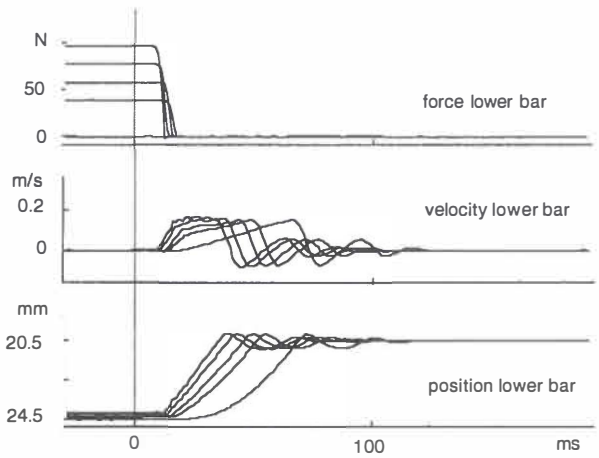


Figure 1B. Control recordings when the apparatus was loaded with 100, 80, 60, 40 or 0 N and the solenoid was switched off by hand (at time 0, dashed line). (Top trace) Force recordings of the lower bar; (middle trace) the (calculated) velocities; and (bottom trace) position recordings.

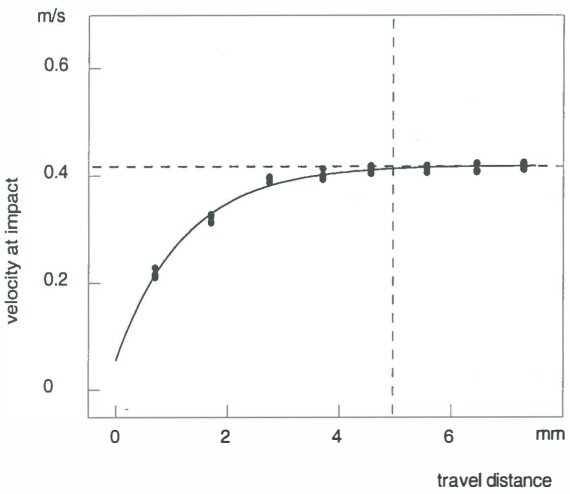


Figure 2. Plot of the impact velocities of the lower teeth/ lower bar combination in 100 N unloading experiments as a function of 8 different travel distances between 0.7 and 7.3 mm (one subject, three trials *per* travel distance, initial mouth opening 24.5 mm). A curve has been drawn between the points, showing that a maximal velocity of 0.42 m/s is reached after a distance of travel of 4.5 - 5 mm; thereafter the velocity becomes constant.

conditions. All experiments were repeated five times, giving a total of 240 observations per subject. The subjects were allowed to familiarize themselves with the experiments and to practice as often as they wished; they were not informed about their performance.

Maximal velocity

In order to establish maximal velocities of the lower teeth/lower bar combination, we recorded from one subject -in 100 N unloading experiments with an initial mouth opening of 24.5 mm- position profiles over 8 different travel distances between 0.7 and 7.3 mm and calculated the velocities at impact. Each experiment with equal conditions was repeated three times, giving a total of 24 observations.

Controls

The mechanical properties of the unloading device were measured by loading it by means of an adjustable clamp exerting forces of 100, 80, 60, 40 and 0 N and switching off the solenoid by hand. These recordings were used as controls. The 0 N controls showed that there was an imbalance of the lower bar, which was measured by adding weights to the bitten end until balance was reached. The imbalance of the lower bar was 4.65 N.

Data analysis

As long as any force was recorded by the strain gauges there was a driving force coming through the lower teeth onto the lower bar, implying that the velocity of the bitten end of the lower bar was the same as that of the lower front teeth. The impact velocity of the lower teeth as a function of initial bite force, mouth opening and distance of travel could be calculated from the position recordings of the bitten end of the lower bar by differentiation of the position signals over time and looking for the maximum. This maximum coincides with impact, because after impact the velocity drops to zero due to a transfer of kinetic energy to the elastic plastic cap covering the adjusting screw in the groundplate of the device.

All processing was done on an Apple

Macintosh IIcx computer system running 'DataMonster 2.0' an analytical program written by one of the authors (E.O). Spearman's ranking test was used to detect possible differences in inter-individual behavior (De Jonge, 1963).

Results

Mechanical properties of the unloading apparatus

Figure 1B gives the output of the lower bar force transducer, the (calculated) velocity of the lower bar, and its position signal when the unloading apparatus was loaded by means of a clamp -with 100, 80, 60, 40 and 0 N respectively- and the solenoid was switched off by hand (at time 0). The initial distance between the bars was 24.5 mm, the distance of travel was 4.5 mm. [Loading the apparatus caused deformation of the upper and lower bar so that -with an initial load of 100 N - the actual distances of travel were 3.7, 2.4 and 1.1 mm in stead of 4.5, 3.0 and 1.5 mm. However, these differences are irrelevant for the interpretation of our results, so that, for reasons of clarity, we will speak of distances of 4.5, 3.0 and 1.5 mm.]

After the current was switched off, it took 11, 12, 14, 16 or 22 ms respectively before the lower bar started to move; at these moments the closing force equalled the declining resistance from the core (which takes some time to disappear due to decaying remnants of the magnetic field in the core). After the lower bar started to move, the force

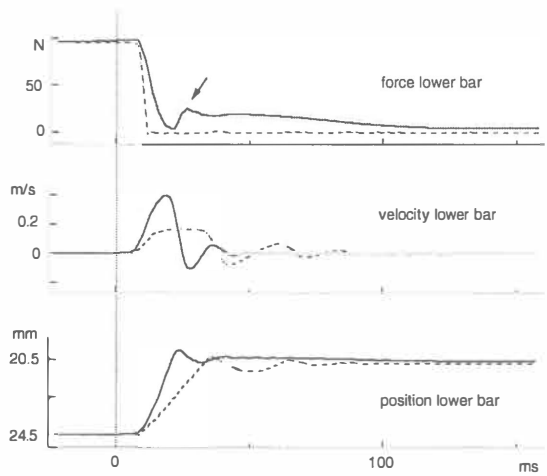


Figure 3. Traces of the average force of the lower bar (thick line upper trace; SD in grey; n=25), the average velocity (middle trace) and the average position (bottom trace) of five subjects (five trials per subject) performing a 100 N unloading experiment at an initial mouth opening of 24.5 mm and a distance of travel of 4.5 mm, and the controls (dashed lines). The average reaction force induced by the teeth pushing on the bar after it was arrested, is indicated by an arrow. At time 0 the solenoid was switched off.

transducer gave output for another 4 to 6 ms before it fell silent; during this time the elastic energy stored in the bars and the clamp was converted into kinetic energy.

The time of travel of the lower bar was 26, 28, 32, 36 or 48 ms, respectively. After the back of the lower bar came into collision with the adjusting screw, a rebound oscillation occurred which was damped in 2 or 3 cycles; the rebound reaction forces can be seen as small humps in the force traces.

In the case of a free fall of the lower bar, the impact velocity over a traveled distance of 4.5 mm was 0.15 m/s.

The experiments

Maximal velocities (0.42 m/s) of the lower front teeth/lower bar combination were reached after a distance of travel of 4.5-5 mm with unloading at 100 N and an initial mouth opening of 24.5 mm; thereafter the velocity became constant (Fig. 2).

Figure 3 depicts traces of the average force of the lower bar (thick line upper trace; standard deviation in grey; n=25), the average velocity (middle trace) and the average position (bottom trace) of the five subjects (each subject five trials) performing a 100 N unloading experiment at an initial mouth opening of 24.5 mm and a distance of travel of 4.5 mm, and the controls (dashed lines). Comparison of the average force and position recording with those of the controls showed that there was always a positive driving force in the experiments, demonstrating that the lower teeth kept contact with the moving lower bar during the entire movement.

Intra-individual variations were small; for each subject within each experimental condition the results were highly reproducible, standard deviations being ≤ 0.02 m/s of the average impact velocities.

Inter-individual variations were also small, illustrated by the fact that recordings from different subjects under equal conditions were highly correlated (Spearman's test, rank correlation coefficient ≥ 0.93). Therefore, we pooled per experimental condition all data and calculated the averages of the impact velocities. The inter-individual variability of the impact velocities, expressed by the coefficient of variation (i.e. average impact velocity/standard deviation) of the impact velocities was between 0.03 and 0.11.

In concurrence with Van Willigen et al. (1997) we found small residual forces after the lower bar was in static conditions again after the impact. The magnitude of these residual forces was comparable to those as described by the abovementioned authors. Since in this study we were primarily interested in the impact velocities of the lower teeth, we did not analyse the characteristics of the residual forces mentioned.

Figure 4A and 4B display the averages of the pooled data (n=25) and their standard deviations of the 24 different unloading conditions, commencing with the greatest (33.5 mm) and with the smallest initial mouth opening (24.5 mm). As can be seen, after unloading the bite force steeply declined (an initial bite force of 100 N declines to 9 N (Fig. 4A) and 3 N (Fig. 4B) within 13 ms after a distance of travel of 4.5 mm).

The relationship between the degree of initial mouth opening and the magnitude of the impact velocity of the teeth is shown of

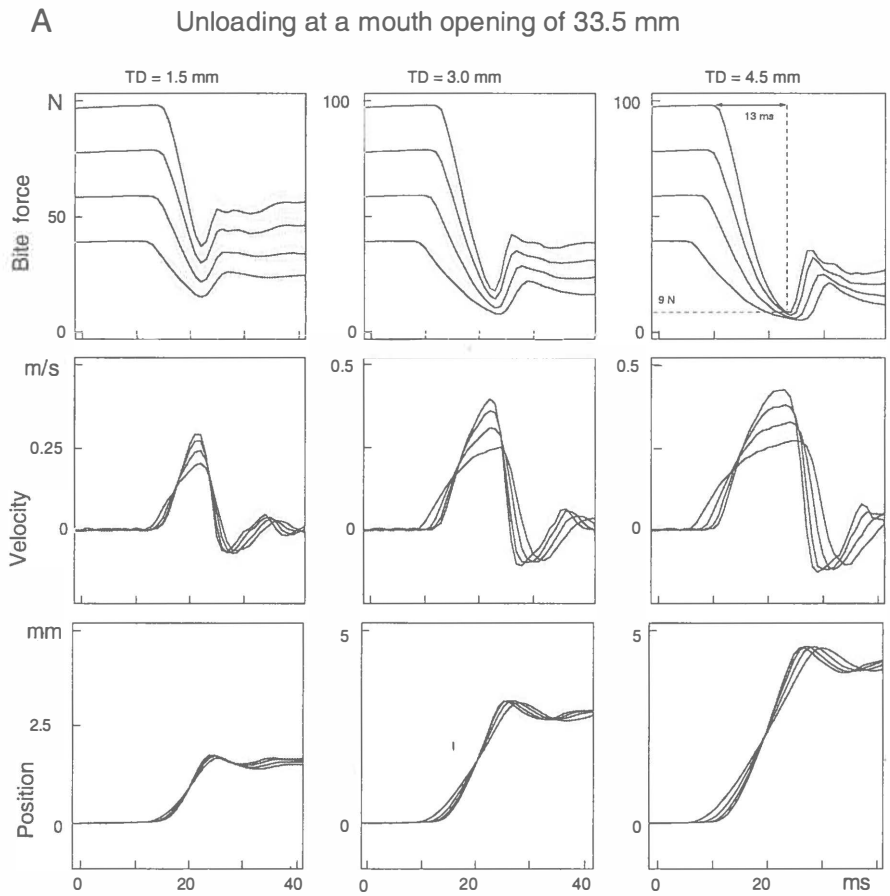


Figure 4A. Summary of the results of 600 experiments (of five subjects, five trials *per* condition, n=25, 12 experimental conditions). The panels depict: forces, velocities and positions of the lower teeth/lower bar combination of unloading experiments at initial bite forces of 100, 80, 60 and 40 N; and initial mouth openings of 33.5 mm at distances of travel (=TD) of 1.5, 3.0 and 4.5 mm.

all experiments with a traveled distance of 1.5 mm (Fig. 5A) and 4.5 mm (Fig. 5B). As can be seen the magnitude of the impact velocity hardly changed when varying initial mouth opening. For instance, at travel distances of 4.5 mm, the impact velocities were 3% (at 80 N) to 8% (at 40 N) larger when starting with a mouth opening of 33.5 mm, as compared to experiments with an initial mouth opening of 24.5 mm.

Figure 6 shows impact velocities as a function of initial bite forces (Fig. 6A) and as a function of distances of travel (Fig. 6B). Since the magnitude of the initial mouth opening had hardly any effect on the impact velocity, we displayed only data from

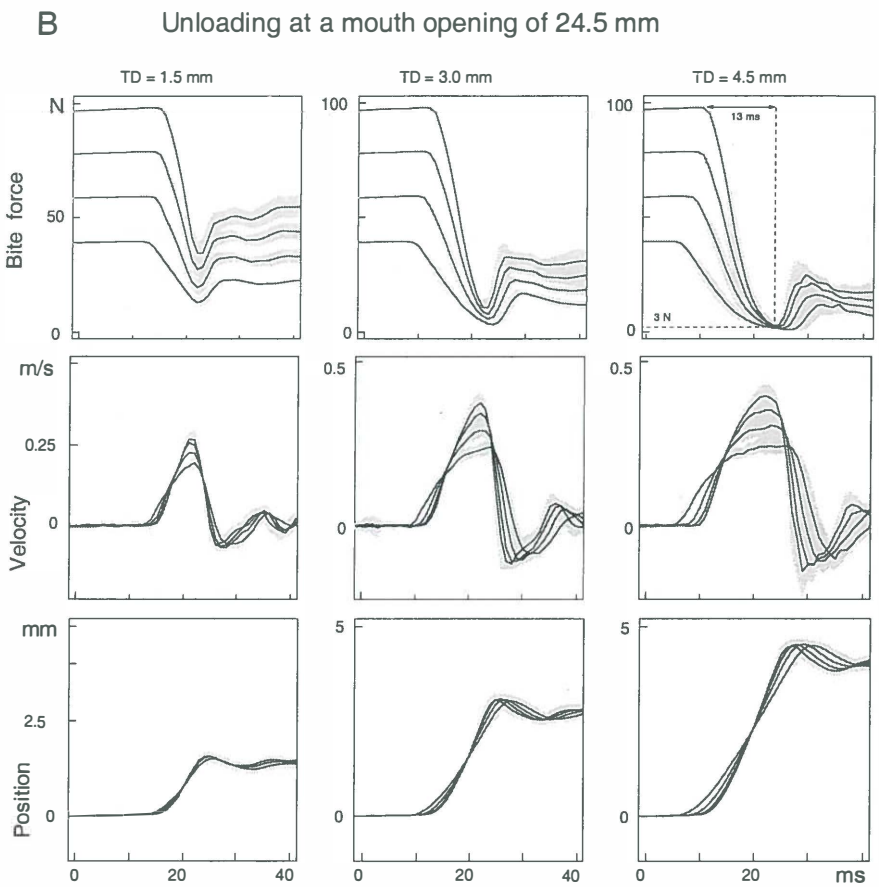


Figure 4B. Summary of the results of 600 experiments (of five subjects, five trials *per* condition, n=25, 12 experimental conditions). The panels depict: forces, velocities and positions of the lower teeth/lower bar combination of unloading experiments at initial bite forces of 100, 80, 60 and 40 N; and initial mouth openings of 24.5 mm at distances of travel (=TD) of 1.5, 3.0 and 4.5 mm.

experiments starting with an initial mouth opening of 24.5 mm. As can be seen from Figure 6B, distances of travel larger than about 3.0 mm gave only little increase in impact velocity, since the bite force had almost vanished after the jaw had moved 3.0 mm (see force traces, Figs 4A and 4B).

Discussion

The observations

In the present unloading experiments, we have shown that the magnitude of impact velocity of the lower teeth after a sudden unloading of a static bite is mainly dependent on the magnitude of the initial bite force

and on the distance of travel of the mandible; it is hardly sensitive to variations in initial mouth opening.

Furthermore, it appeared that the maximal velocity of the lower teeth is small; for example, the maximal velocity is only 0.43 m/s at an initial bite of 100 N, which is reached after a distance of travel of about 4.0 mm. Thereafter it becomes constant.

Impact velocity

In our experiments, the impact velocity of the lower teeth/lower bar combination is determined by both the mechanical properties of the unloading device and the biomechanical properties of the jaw system and its muscular activity.

Considering the dynamic properties of the unloading device, the most important factor is the moment of inertia of the lower bar (i.e. that property of the lower bar that gives the bar resistance against a rotational acceleration). When a (bite) force on the lower bar suddenly lacks a counter force (from the solenoid), it accelerates the bar. Due to the decaying remnants of the magnetic field in the core of the solenoid, this counter force does not disappear instantaneously; this effects the build up of the velocity of the bar. Also, the lower bar, by losing its resistance at the back, makes a sweeping motion, influencing the dynamics of its movement. Moreover, there is a small imbalance of the lower bar, in favor of a closing movement.

All these factors determine the resistance of the device. However, in view of the large differences in dynamics in the unloading experiments, as compared to those of the control experiments (Fig. 3), it is clear that the closing force exerted by the jaw muscles is the predominant factor in pro-

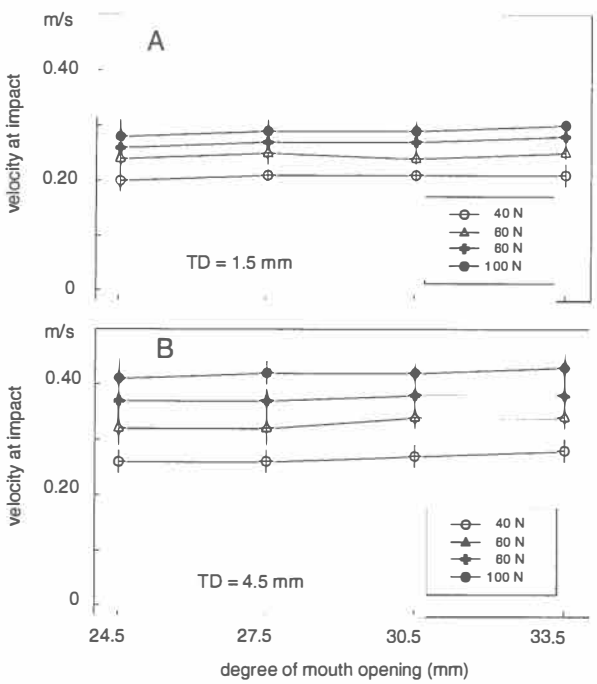


Figure 5. Averaged impact velocities of the lower teeth/lower bar combination and their standard deviations after a sudden unloading (n=25, SD ≤ 0.04 m/s), as a function of the initial mouth opening, at 4 initial bite forces and distances of travel of 1,5 mm (Fig. 5A) and 4,5 mm (Fig. 5B). Note that the magnitude of the impact velocity hardly changes when varying the initial mouth opening. TD = traveled distance.

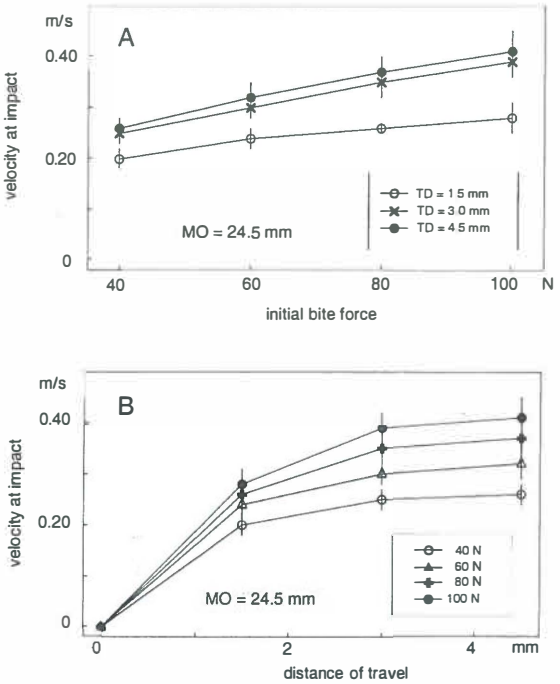


Figure 6. Averaged impact velocities of the lower teeth/lower bar combination and their standard deviations after a sudden unloading (n = 25, SD ≤ 0.04 m/s) as a function of the initial bite force (Fig. 6A) and of the distance of travel (Fig. 6B). Since the impact velocity is hardly sensitive to variations in the initial mouth opening, the results are shown at an initial mouth opening of only 24.5 mm. As can be seen, the impact velocity depends both on the initial bite force and the distance of travel. TD = traveled distance, MO = initial mouth opening.

ducing the dynamics of the movement.

The impact velocity of the lower front teeth appeared to be largely dependent on the initial bite force and its steep decline after unloading (Fig. 4); this steep decline in force is the cause for the limitation of the impact velocity we observed. If the biting force would not decline and stay at a level of 100 N, a 0.20 kg mass (approximately equal to the effective mass of the lower jaw and bar together) would reach a velocity of 1.9 m/s over a distance of 4.5 mm. This is about 340 % higher than the velocity calculated from our experimental results, and equivalent to a 1850 % higher impact energy. [If we define F to be the constant force, m to be the mass, a(t) to be the acceleration, v(t) to be the velocity and s(t) to be the position, we obtain the following expressions: Since F = m.a it follows that a = F/m and by integration $v(t) = (F/m).t$ and by further integration $s(t) = 0.5\ (F/m).t.t$ so that travel time is $t = \sqrt{(s/(0.5\ F/m))}$. At a distance of 0.0037 m (i.e. accounting for deformations of the bar) and a mass of 0.20 kg, the travel time of the mass, driven by a force of F = 100 N, is $t = \sqrt{(0.0037 / (0.5 * 100/0.2))} = 0.0038$ s and the impact velocity $v(0.0038) = 0.0038 * 100 / 0.2 = 1.9$ m/s.]

Possible explanations

Since neural control is too slow to explain the quick fall in force (see 'introduction'), the steep decline in bite force can only be explained by the force-length and force-velocity properties of the contracting jaw-closing and co-contracting jaw-opening muscles; these properties are both generated by activation of the muscles.

Force-length properties

In the present study, we have shown that the impact velocity hardly is sensitive to variations in degree of mouth opening. This is due to the stereotyped way the force declines after unloading at these mouth openings (compare Fig. 4A with 4B).

The active force-length curves of the closing muscles can only be responsible for this decline if they have a rising, constant and very steep slope at mouth openings between 20 and 33.5 mm. This can not be the case, since Mackenna and Türker (1983) showed in humans a maximal closing force at about 17 mm mouth opening, which varied hardly between 14 and 20 mm. This indicates that at an initial mouth opening of 20 mm or greater, muscle fibers will be on the descending part of the force-length curve. Consequently, active force-length properties of the closing muscles can be ruled out from being responsible for the steep decline in bite force and thus for limiting impact velocity.

Furthermore, Van Willigen et al. (1997) showed in experiments comparable to ours, that an initial bite force of 100 N -unloaded at an initial mouth opening of 19 mm- has vanished after a jaw travel of about 6.5 mm and remains absent when the bar has come to a standstill. In this situation the teeth are still far from occlusion. If force-length properties of the jaw-closing muscles would underlie this phenomenon, this would imply that no force is left when the teeth come in occlusion, which is in contradiction with everyday experience.

It is also unlikely that force-length properties of the opening muscles are of much help in reducing the bite force, since this would require a high force production of the jaw-opening muscles and a muscle architecture with sufficiently short muscle fibers. This is neither in agreement with the magnitude of co-contraction of the opening muscles observed [which is in general very low (Miles and Wilkinson, 1982; Van Willigen et al. 1997)] nor with the observed architecture of human mouth opening muscles (T.M.G.J. Van Eijden, personal communication).

Force-velocity properties

Otten (1991) and Slager et al. (1995) attribute the steep decline in bite force to the activation dependent force-velocity properties of both the jaw-closing and -opening muscles. This fits nicely with our observation that the impact velocity of the teeth is hardly sensitive to variations in initial mouth opening, since force-velocity effects are largely independent of muscle length and thus of mouth opening. Secondly, we found that the percentage of loss in force is independent of the magnitude of the initial bite force (Fig. 4). This suggests that the decline in force is activation dependent as are force-velocity properties.

When the mandible came in static circumstances again after the jaw system was arrested, there was a residual bite force left. This residual force was so small that its magnitude cannot be explained by neural control and/or bio-mechanical properties of the functioning muscular ensemble alone. This phenomenon will be subject to further study.

Force-velocity properties are helpful in avoiding teeth damage

By exclusion, we conclude that the force-velocity properties of the contracting jaw muscles are mainly responsible for the decline in bite force, and thus for a limitation of impact velocity after a sudden unloading of the jaw. This is of great significance for the oral system, since a reduction of impact velocity of the teeth by a factor of two corresponds to a reduction of impact energy by a factor of four, since the kinetic energy of the jaw is proportional to the square of its velocity. In such a situation, the activation dependent mechanical properties of the jaw muscles are helpful in avoiding damage.

This also holds for other motor tasks in which neural processing is too slow to handle instances of unexpected and undesired load perturbations. Therefore, we hypothesize that the force-velocity properties of active muscles are useful in handling unexpected perturbations during movement at a very early stage.

Acknowledgements

The authors are grateful to Mr. J. Mast and Mr. K. Vaartjes for designing and building the unloading apparatus and for their help during the experiments.

3

A mathematical model of the human jaw system simulating static biting and movements after unloading

Summary

When the resistance to a forceful isometric bite is suddenly removed in unloading experiments, the bite force drops to zero and the mandible reaches a constant velocity. This occurs at an initial bite force of 100 N after about 12 ms when the incisors have moved 4.5 mm. Reflex activity is far too slow to limit the velocity at impact. To explore the influence of other factors (co-contraction, force-length properties and force-velocity properties of the muscles) on the velocity at impact, a numerical forward dynamic model of the jaw system is formulated. Unloading experiments in different experimental conditions were simulated with the model. Most parameter values of the model are based on physiological data, both from literature and a data basis from a human cadaver study. Other parameter values were found by optimally fitting the model results to data from the unloading experiments. The model analysis shows that the limitation of the jaw velocity mainly may be due to the force-velocity properties of the jaw-closing muscles. Force-length properties of the jaw muscles hardly contribute to the impact velocity. The compliance of tendinous sheets in the jaw muscles is unfavorable for the limitation in impact velocity, whereas co-contraction of jaw-opening and -closing muscles helps to limit impact velocity. The force-velocity properties of the muscles provide a quick mechanism for dealing with unexpected closing movements, and so avoid damage to the dental elements.

Introduction

The human oral system is so powerful, that it can easily damage its dental elements. Biting through hard and brittle food especially can result in high impact velocities of the dental elements. Any mechanism that reduces the bite force as soon as a movement commences may prevent damage to the teeth, because the impact velocity will be lower.

In unloading experiments (in which the resistance to a forceful static bite is suddenly withdrawn experimentally), it is shown that the bite force does indeed decrease at high rate as soon as the mouth starts closing (Miles & Wilkinson, 1982; Van Willigen et al., 1997). After the bite force vanishes after about 12 ms (Nagashima et al., 1997), the velocity no longer grows, because by virtue of Newton's law the velocity of a solid body is constant when no forces are exerted on the body. The aim of the present study is to uncover the factors that may cause this quick decrease in bite force and the limitation in the velocity of the mandible.

Reflex events can be excluded because these events occur too late (Hannam et al., 1968; Lamarre & Lund 1975; Miles & Wilkinson 1982; Van Willigen et al., 1997; Yoshida & Inoue, 1995) to have any significant influence on control of the movement of the mandible.

Co-contraction of the jaw-opening and -closing muscles might be a factor that can cause a quick drop in force. Miles & Wilkinson (1982) suggest that the resistance to elongation of the activated co-contracting digastric muscles (resulting in an increase in jaw-opening force) is possibly responsible for the quick decrease in bite force and the limited mandibular velocity. They suggest that the resistance is perhaps due to distortion of cross bridges between myofilaments (Rack & Westbury, 1974). However, Van Willigen et al., (1997) show that various levels of co-contraction have hardly any influence on the decrease in bite force during mouth closure.

Also the force-length properties of muscles can play a role in the drop in bite force. Slager et al. (1995) show in a model study (with linearized muscle models, not based on morphometric data) that the quick decrease in bite force can be attributed partly to the force-length characteristics of the jaw-closing and -opening muscles.

Furthermore, the force-velocity properties of the jaw muscles can have profound effects on the dynamic bite force (Otten, 1991; Slager et al., 1995): the jaw-closing muscles can lose a fair amount of their force when they shorten, and the opening muscles can gain force when they are stretched, resulting in vanishing of the bite force.

However, from unloading experiments only, it is hard to judge how co-contraction, force-length, and force-velocity properties contribute to the decline in bite force during jaw movement (especially because the length and the velocity of the muscle fibers is unknown, due to compliance of tendinous sheets). Numerical models can serve as a tool to find their relative contribution.

With this in mind, we have formulated a mathematical model that can simulate unloading experiments. By removing the force-length or force-velocity properties

of the jaw muscles, the compliance of the tendinous sheets, or by changing the level of co-contraction of the jaw muscles, their contribution to the dynamic bite force and thus to the magnitude of the impact velocity can be studied.

Material and methods

Introduction

We have formulated a mathematical forward dynamic model of the jaw system that can simulate jaw unloading experiments. For that we needed

1) a model of the unloading device ('*device model*') with which experiments were done.
2) muscle models of the jaw-closing muscles (hereafter, '*muscle model 1*') and jaw-opening muscles ('*muscle model 2*'), based upon morphological, physiological, and biomechanical properties.
3) to tune the model, we used results (force profiles, positions, and velocities of the mandible) from the unloading experiments, described in chapter 2 (hereafter, 'the experiments').

The experimental set-up can be summarized as follows. An 'unloading' device (Fig. 4A) was used comprising two parallel aluminum bars, of which the upper was attached to two vertical plates mounted on a base plate. The lower bar hinged around an axis. Bite forces were exerted between the upper and lower incisors and cuspids on both bars of the device. For recording bite forces, strain gauges were attached on the lower bar. The initial resistance to closing was achieved by an empowered solenoid. The solenoid could be switched off -triggered by the output of the lower bar strain gauges- at a voltage equivalent to 100, 80, 60, and 40 N (hereafter 'initial bite force'). When the solenoid was switched off, the lower bar dropped at the back, and its bitten end was lifted up. The displacement of this bitten end was measured. The distance of travel of the lower bar could be varied by means of an adjusting screw. To buffer the shock of collision, this screw was covered with a rubber cap. To vary the initial mouth opening, the position of the upper bar could be adjusted by means of slotted holes and bolts.

Five participants were asked to bite without visual guidance through a resistance of 100, 80, 60, or 40 N with care. This was done at four initial mouth openings (24.5, 27.5, 30.5, and 33.5 mm) and three distances of travel of the lower bar (1.1, 2.4, and 3.7 mm), giving 48 different experimental conditions. The mouth opening (MO) of the participants was defined as the interincisor distance added to the vertical overlap of the dentition (overbite). All experiments were repeated five times per subject. We used the data of these experiments.

Model of the jaw system

The model of the jaw system calculates forces, velocities and positions of the mandible as a function of the initial bite force, mouth opening, and distance of travel of the mandible after the moment of unloading.

Figure 1 illustrates a diagram of the model of the jaw system. As can be seen, the model is built up from three compartments:

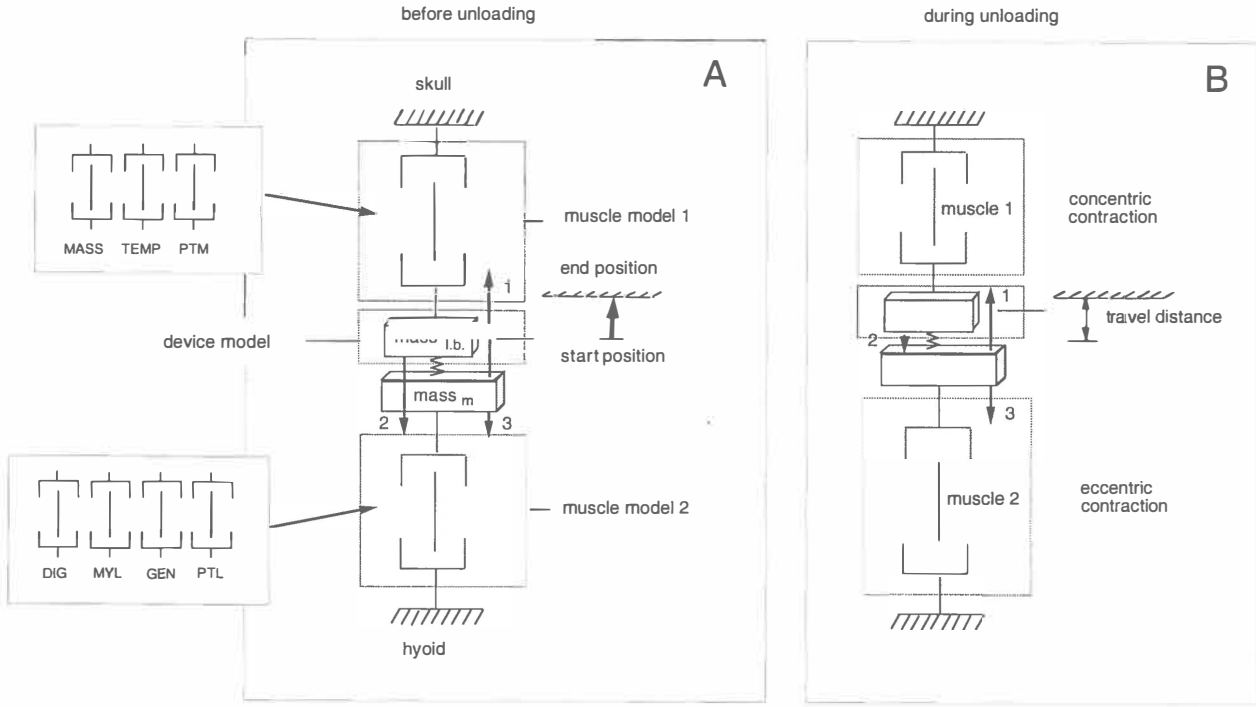


Figure 1. Diagram of the model of the jaw system used for simulating the unloading experiments as described in chapter 2. Forces involved are coded as: 1, $F_{closers}$; 2, $F_{resistance}$; 3, $F_{openers}$. Unloading starts with a quick reduction of $F_{resistance}$ (A). Result is an acceleration of the mandible (with $mass_m$) and the lower bar (with $mass_{lb}$) of the unloading device (see Fig. 4A), accompanied by a concentric contraction of the jaw-closing muscles and an eccentric contraction of the jaw-opening muscles (B).

1) MUSCLE MODEL 1. All the jaw-closing muscles (the masseter, temporalis and medial pterygoid muscles), together were modelled as one single muscle (muscle 1), attached to the fixed skull and to the mandible. The mandible is attached to the lower bar of the unloading device by means of a stiff spring, simulating the suspension of the dental elements.

2) MUSCLE MODEL 2. The jaw-opening muscles (the digastric, mylohyoid, geniohyoid and lateral pterygoid muscles) were also modelled as a single muscle (muscle 2), attached to the fixed hyoid bone and to the mandible.

3) DEVICE MODEL. The resistance of the unloading device was modelled by a suitable set of mathematical formulae, adequately describing the measured properties of the device (see Device model).

Apart from the intramuscular movements, the model of the jaw system has only one degree of freedom; i.e. the movement of the lower bar and mandible along the line of action of both muscles. [This is in contrast with the work of Laboissière et al. (1996), who have produced a model of the jaw system with seven muscles with separate jaw and hyoid bone movements. Such a sophisticated model is useful in the context of the study of multimuscle control systems, but would be out of place in our more limited scope.]

The line of action of our model has a direction that is perpendicular to the occlusal plane and passes through the canines. (In reality the jaw muscles have a distributed attachment to the lower jaw, which has a variable center of rotation. To keep the model simple and yet realistic, we calculated the ratios between the force produced by the jaw muscles and the resultant force at the lower canines. The resultant force was used, because this way we could compare directly the external measured forces in dynamic equilibrium with the model output.)

The mentioned ratios are the kinematic transmissions from the muscles to the teeth. The actual muscles then could be described by two model muscles, which have properties that compensate for the simplified geometrical arrangement of the model.

The forces produced by muscles 1 and 2 were called: $F_{closers}$ and $F_{openers}$. Because the net muscle force produced by opening and closing muscles together is consumed partly by acceleration of the mandible, the force measured on the lower bar (F_{output}) is less than the total net muscle force. We therefore defined

$$F_{output} = F_{closers} - F_{openers} - mass_m \cdot acc_m$$

in which $mass_m$ is the mass of the mandible and acc_m is the acceleration of the mandible. The resistance of the unloading apparatus as calculated by the device model was called $F_{resistance}$ and the initial bite force was called F_{start}.

At the start of the simulation, the modelled muscles contract isometrically, exerting a bite force equal to F_{start}, so that

$$F_{closers} - F_{openers} = F_{start}, \quad \text{and}$$
$$F_{start} = F_{resistance} = F_{output}.$$

After time $t = t_{unloading}$, $F_{resistance}$ drops quickly, resulting in an acceleration of the lower bar (with $mass_{lb}$) and the mandible

(with $mass_m$) accompanied by a concentric contraction of muscle 1 and an eccentric contraction of muscle 2 (Fig. 1B).

The model of the jaw system simulates a short static phase before unloading and the dynamic phase after unloading until the pre-set distance of travel is reached. In the simulations, the lower bar of the device and the mandible were described as separate solid bodies with masses. When we simulated the behavior of the oral system in free motion, we removed the mass and resistance of the device from the model.

Figure 2 offers a flow diagram of the simulations. The initial conditions of the model simulations were: F_{start} and $F_{openers}$ (see APPENDIX: tuned parameters), the initial mouth opening, and the travel distance of the lower bar.

We calculated F_{output} and the velocity and position of the lower bar as follows: $F_{closers}$ was calculated by

$$F_{closers} = F_{start} + F_{openers}.$$

We derived $F_{resistance}$ from the device model (see Device model). Because $F_{output} = F_{closers} - F_{openers} - mass_m . acc_m$, we could define the driving force on the lower bar as

$$F_{drive} = F_{output} - F_{resistance}.$$

The acceleration of the solid body representing the lower bar could be calculated

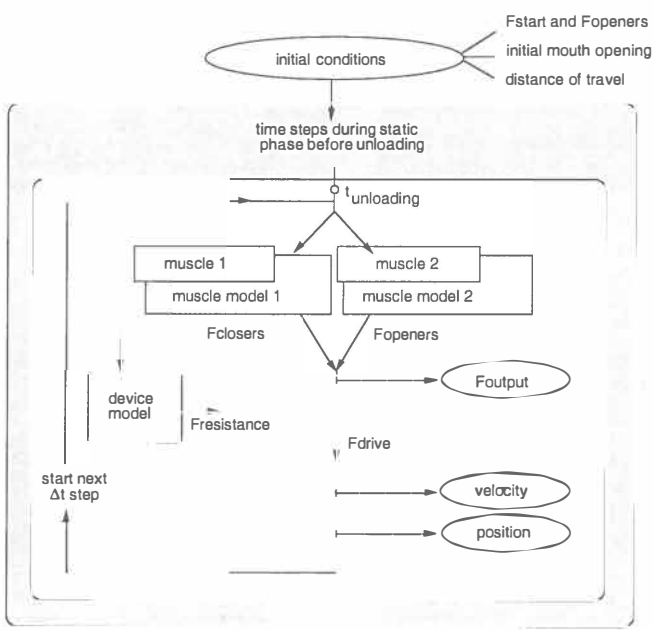


Figure 2. Flow diagram of the model. Input of the model are the initial conditions: the initial bite force (F_{start}) and force of muscle 2 ($F_{openers}$), the initial mouth opening and the distance of travel. Output of the model are the bite force (F_{output}), the velocity and the position of the lower bar.

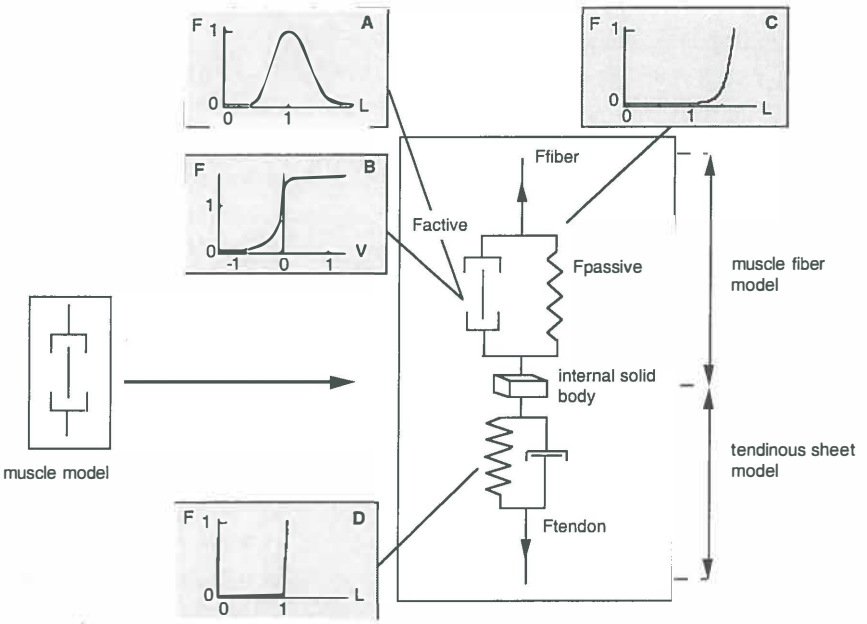


Figure 3. Diagram of the muscle model. Muscle model contains a model of lumped muscle fibers, a model of a tendinous sheet and a small internal solid body. Active force (F_{active}) is calculated from the recruitment, fiber length (curve A) and fiber contraction velocity (curve B). Passive force ($F_{passive}$) is calculated from the strain of the passive structures of the muscle fibers (curve C). Force exerted by the muscle fibers (F_{fiber}) is equal to $F_{active} + F_{passive}$. Force exerted by the tendinous sheet (F_{tendon}) is dependent on its strain (curve D).

from this F_{drive} and its mass. From this, by numerical integration, we were able to calculate the velocity and position of the lower bar and mandible.

Numerical integration with time steps of 0.1 ms allowed accurate calculation of the changing time-, position-, and velocity-dependent forces ($F_{closers}$, $F_{openers}$, and $F_{resistance}$). The recruitment of muscle 1 and muscle 2 was calculated in the first time step from F_{start} and $F_{openers}$ by inverting Otten's (1987a) formula for the activation dynamics of muscle fibers, producing muscle force from recruitment. The recruitment of both modeled muscles was kept constant throughout the whole simulation.

Muscle model

Muscle models 1 and 2 were founded on the muscle model written by Otten (1987a); a model of a tendinous sheet was added; Figure 3 gives a diagram.

Each muscle model contains a model of lumped muscle fibers, a model of a tendinous sheet and a small internal solid body (with a mass) representing the effective moving muscle tissue.

The force exerted by the contractile part of the muscle model (F_{fiber}) is the resultant of *active* forces (F_{active}) [calculated from the recruitment, fiber length (curve A) and fiber velocity (curve B)] and *passive* forces

($F_{passive}$) [depending on the strain of the muscle belly (curve C)]. The force exerted by the tendinous part of the muscle model (F_{tendon}) depends on the strain of the tendinous sheet (curve D).

Note that the length and the velocity of the contractile part of the muscle model depends on the position and velocity of the mandible and the length and rate of length change of the tendinous sheet.

Because the contractile part and the tendinous part of the model are positioned in series (Fig. 3), the force exerted by the muscle model equals F_{fiber} and equals F_{tendon} before unloading. After unloading, however, F_{fiber} - F_{tendon} becomes non-zero, so that the internal solid body is accelerated. To avoid intramuscular oscillations we added damping properties to the tendinous part of the model, in line with Hannam and Langenbach (1995).

Muscle models 1 and 2 differ in the values of their morphometric and muscle parameters (see Parameters used in the muscle model).

Device model

The purpose of the device was simply to quickly let the resistance to the bite decline in a reproducible way: no attempt was made to perform any servo tracking of the resulting movement.

The resistance offered by the device ($F_{resistance}$) after unloading appeared to be a complex ensemble of force components. After recording the resistance of the device in a large collection of tests, a search procedure was started to find the parameters of a set of mathematical formulas describing these force components (see Device parameters, APPENDIX).

Four force components can be discerned:

F_{offset} (the resultant of dry friction between the lower bar and apparatus and an imbalance of the lower bar),

F_{magnet} (being the decaying remnants of the magnetic field),

$F_{vibration}$ (a time varying force originating in a damped vibration of the lower bar) and

F_{end} (formed by the first contact of the lower bar with its rubber rest) apart from the force generated by accelerating the lower bar of the device. Figure 4B gives an example of a model simulation of $F_{resistance}$ (thick curve) and its force components (initial loading 100 N; travel distance 3.7 mm).

Parameters used in the models

The parameters used in the muscle models can be subdivided into three sets: parameter values from literature (Table A1), morphometric parameter values taken from a cadaver study by one of us (Van Eijden) (Tables A2 and A3), and parameters of which only rather wide ranges were found in literature. The values of the last set of parameters could be found by tuning of the model (Table A4A). For the value of the passive damping of the tendons we have chosen a specific value (Table A4B). The parameters are described in the APPENDIX.

Sensitivity analysis

In order to develop some notion on the relative meaning of model parameters for the force output, we performed a sensitivity analysis by testing the effect of an increase of 1% of the value of each parameter separately on the percent change in force output

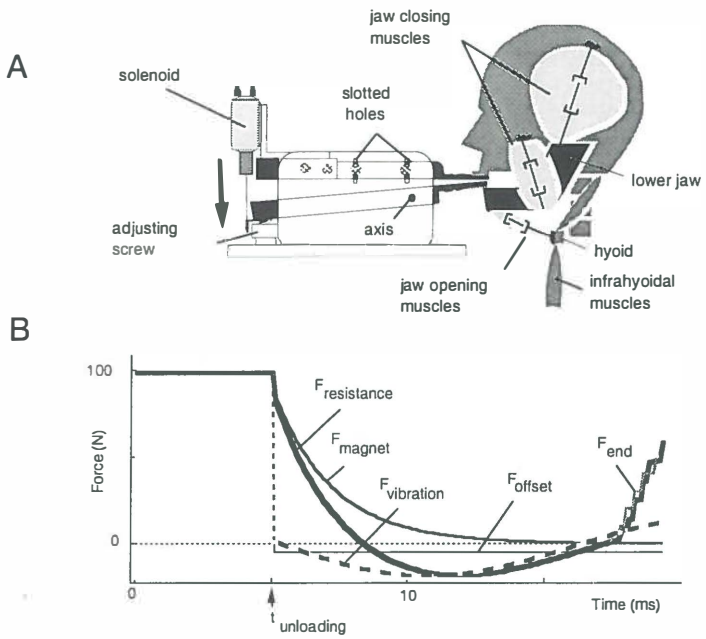


Figure 4. (A) Unloading device: 2 parallel aluminum bars (with pairs of strain gauges) are attached to 2 metal plates mounted on a base plate; the lower bar is fixed to an axis. Initial resistance to closing is achieved by a solenoid. Solenoid could be switched off at voltages equivalent to 100, 80, 60 and 40 N. (B) An example of a model simulation of the unloading device at an initial loading force of 100 N and a distance of travel of the lower bar of 3.7 mm. It shows the counter force produced by the unloading device ($F_{resistance}$, thick line). Before unloading, $F_{resistance}$ is equal to the initial loading force. After unloading, $F_{resistance}$ rapidly declines, its profile being the sum of F_{offset}, F_{magnet}, $F_{vibration}$, and F_{end}.

of the model.

Search procedure

After setting of the morphometric parameters and the literature determined parameters in the model, a search procedure was started to optimize parameter values by obtaining a minimal least squares fit of the model results on the experimental data (see APPENDIX).

RESULTS

Comparing model output with experimental results

Figure 5 shows the fit of the model simulations (thick lines) with the experimental data (thin lines, SD in grey, n=25) of eight experimental conditions. The panels depict forces, velocities, and positions of the lower bar at initial forces of 40 and 100 N, mouth openings of 24.5 and 33.5 mm, and travel distances of 1.1 and 3.7 mm. A comparison

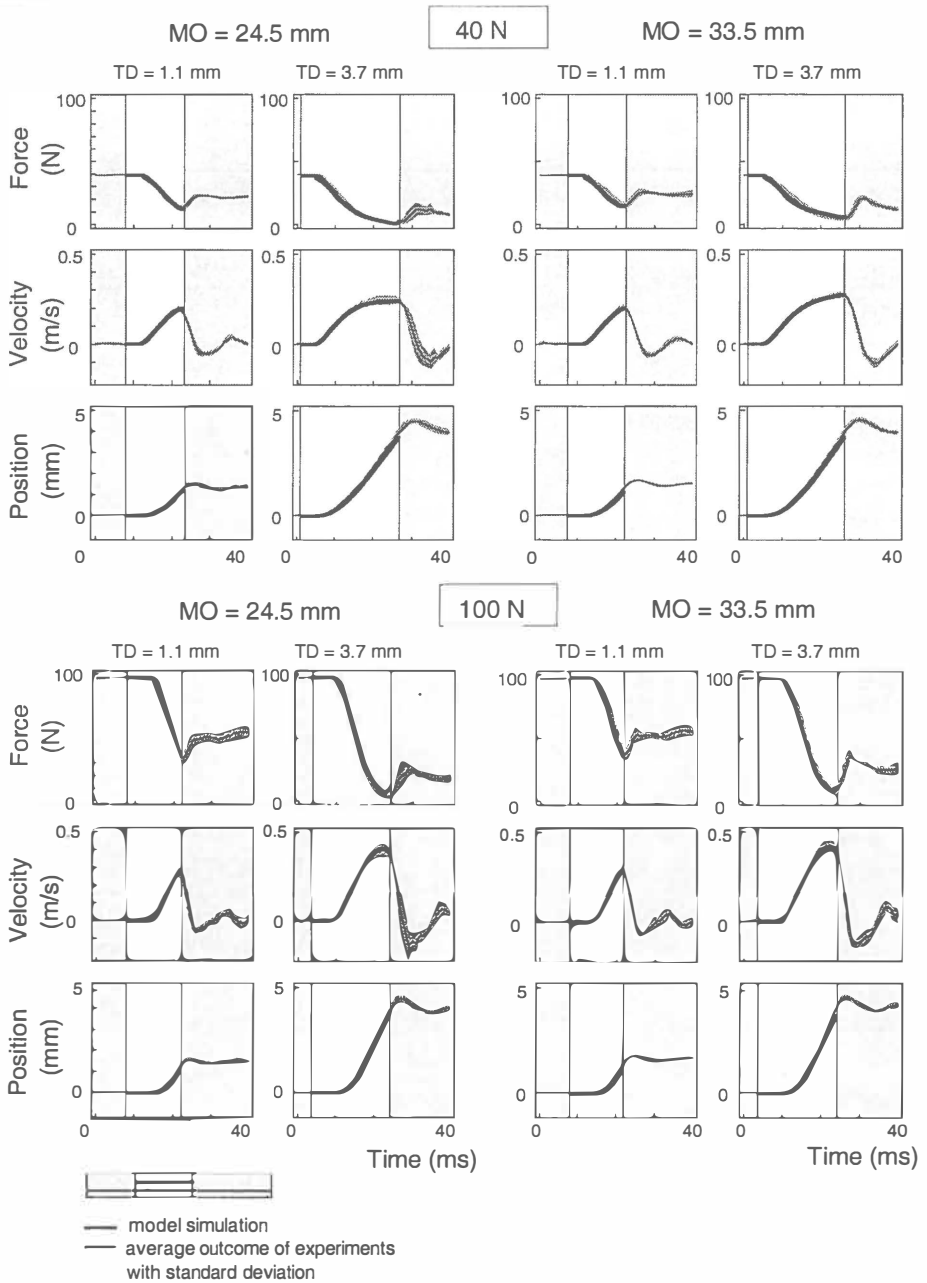


Figure 5. Results of 8 groups of experiments, as described in chapter 2, and their model simulations. Depicted are forces, velocities, and positions of the lower bar (thin lines, experimental results with SD in grey bands; thick lines, model results) at an initial bite force of 40 and 100 N, a mouth opening (MO) of 24.5 and 33.5 mm, and a distance of travel (TD) of 1.1 and 3.7 mm. Model results are only depicted in the white area's in each panel in the figure.

is made from the time of unloading until the pre-set distance of travel was met by the model (white time blocks).

As can be seen, the model output is in good agreement with the experimental results, which also is illustrated in Figure 6. The latter figure gives impact velocities as calculated by the model plotted against the measured average impact velocities of the experiments. As can be seen the data lie close to the line of equality.

Factors contributing to the reduction in bite force after unloading

Figure 7 gives information on the internal mechanics of muscle 1 and 2 in one simulation of an unloading experiment of 100 N (mouth opening 33.5 mm, travel distance 3.7 mm). It shows the time course of forces, lengths, and velocities of various components of muscles 1 and 2. The figure serves as an example. In the following text, numerical references will be made to other conditions which have not been included in Figure 7.

Figure 7A illustrates for the jaw-closing (top) and jaw-opening muscles (bottom) the contribution of F_{active}, $F_{passive}$, $F_{closers}$, and $F_{openers}$ to F_{output}. From Figure 7A, top, it can be seen that before unloading $F_{closers}$ mainly consists of active muscle force (82%). [At a smaller initial bite force (40 N) at the same mouth opening, the active force of muscle 1 takes up a much smaller part of $F_{closers}$ (52%).] The passive force of muscle 2 (bottom) is almost zero, so that muscle 2 delivers active force only.

After unloading, the bite force (F_{output}) drops from 98 to 9 N within 13 ms. Looking at the contribution of the different components, the active force of muscle 1 decreases rapidly from 84 to 4 N, whereas its passive force decreases from 19 to 15 N. At the same moment the active force of muscle 2 increases from 5 to 12 N. Furthermore, 2 N is produced by deceleration of the lower jaw. Apparently, the quick reduction in F_{output} is caused mainly by the sharp decrease of the active force of muscle 1 (closing muscles), whereas the changes in passive force of muscles 1 and 2 hardly contribute to the effect.

Figure 7, B and C, illustrates the relative length and velocity of muscles 1 and 2, and of their muscle fibers and tendinous sheets. The length of the structures is set to zero at the start of unloading to make comparison easy.

It can be seen that after unloading the changes in length and velocity of the muscle fibers are much smaller than that of the

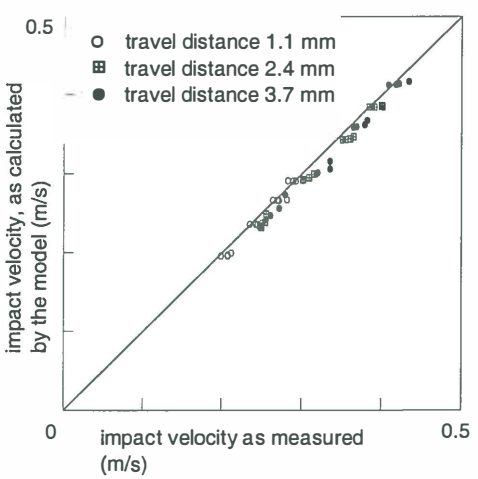


Figure 6. Impact velocities calculated by the model plotted against the averaged impact velocities as measured in the 48 experimental conditions (described in chapter 2).

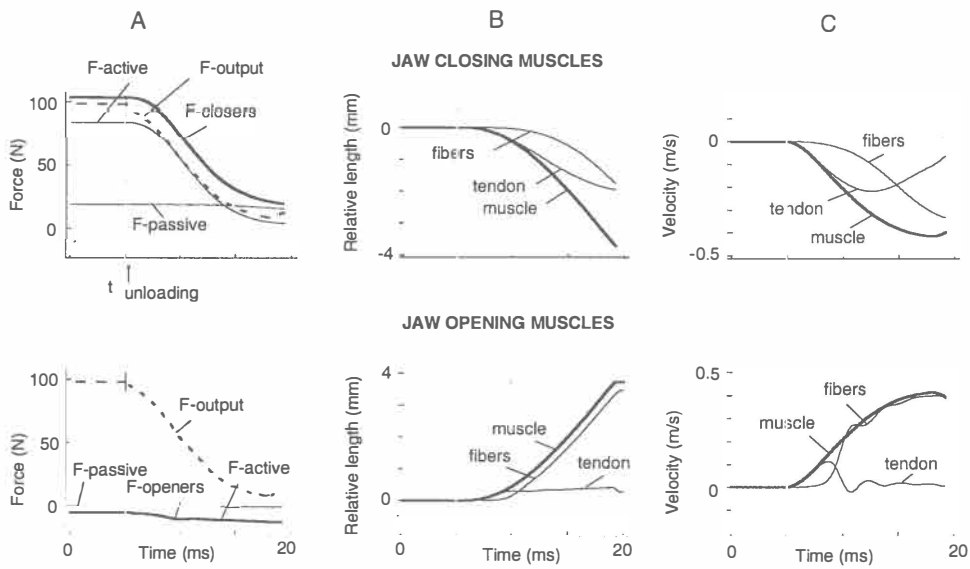


Figure 7. Data on the internal mechanics of muscles 1 and 2 in a simulation of an unloading experiment of 100 N (initial mouth opening 33.5 mm, distance of travel 3.7 mm). Time course of various forces (i.e., the contribution of F_{active}, $F_{passive}$, $F_{closers}$, and $F_{openers}$ to F_{output}), the lengths and the velocities of the components of muscles 1 and 2 are shown. (A) Note that the quick reduction in F_{output} is caused mainly by the sharp decrease of F_{active} of muscle 1, representing the closing muscles. (B) and (C) Note that after unloading, the changes in length and the velocity of the muscle fibers is much smaller than that of the muscle.

muscle. [At a change in length of 3.7 mm of muscle 1, the length change of the muscle fibers is only 1.8 mm (48%)]. This is due to the in series arrangement of the tendinous sheet and the fibers, and to differences in their mechanical properties. After unloading, the tendinous sheet and the fibers suddenly shorten with the tendon initially taking up most of the slack length (≤ 52%). At the end of the movement, the length of the tendinous sheet increases again, whereas the muscle fibers still shorten. A similar effect, though more short lasting, can be seen in muscle 2.

Figure 8 shows the force-length (Fig. 8A) and force-velocity properties (Fig. 8B) of muscles 1 and 2. The ranges covered during a simulated experiment also are shown (conditions: 100 N initial force, 33.5 mm mouth opening and 3.7 mm travel distance). Black symbols indicate the start of the experiment, whereas white symbols illustrate its end. Normalized length is defined as the length of the muscle divided by its optimal length, which is the length at which the muscle produces its highest isometric active force.

The figure illustrates that after unloading the fibers of muscle 1 shorten from a normalized length of 1.22 to 1.19, whereas those of muscle 2 lengthen from 0.71 to 0.78. (At a mouth opening of 24.5 mm and the same travel distance these figures are 1.10 to 1.08 for muscle 1, and 0.91 to 0.98

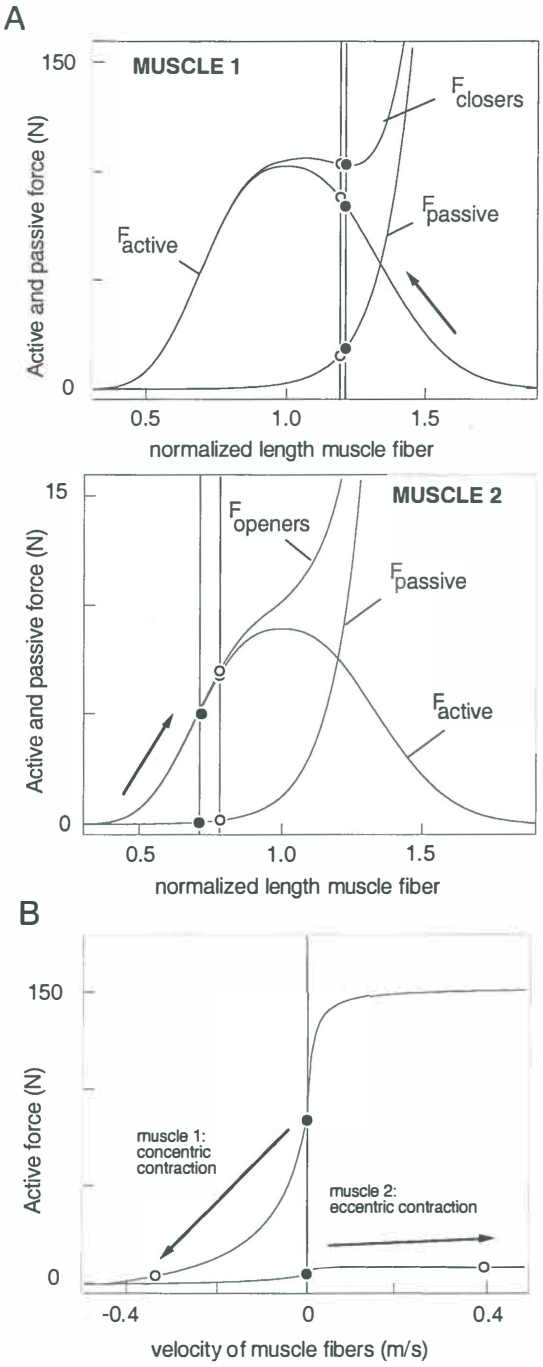


Figure 8. Influence of the active and passive force-length properties (A) and force-velocity properties (B) of the fibers of muscles 1 and 2 on the bite force in a simulated unloading experiment (initial bite force, 100 N; initial mouth opening, 33.5 mm; distance of travel, 3.7 mm).

(A) It can be seen that, because of their force-length properties, muscles 1 and 2 showed a small increase in force during the dynamic phase after unloading.

(B) Because of the force-velocity characteristics of the muscle fibers, F_{active} of the concentric contracting fibers of muscle 1 dramatically decreases, whereas F_{active} of the eccentric contracting muscle fibers of muscle 2 increases marginally in its absolute value.

for muscle 2). The relative change in fiber length of muscle 2 is larger (7%) as compared with that of muscle 1 (2-3%). This is due to the fact that muscle 2 has a shorter length than muscle 1, and that there is less tendon creep of muscle 2 due to the muscles low activation.

It appeared that in all 48 experimental conditions simulated -due to their active force length properties- muscle 1 as well as muscle 2 showed a small increase in active force when the force velocity effects are disregarded (the isometric active force). At an initial force of 100 N and a travel distance of 3.7 mm, the increase in isometric active force of muscle 1 is between 1.2 and 4.1 N and between 0.1 and 1.9 N of muscle 2. These values are too small to contribute significantly to the large reduction in bite force after unloading.

Note that the passive force-length properties of muscle 2 have no influence on the model results, because muscle length was well below the rising part of the force-length curve.

From the force-velocity relationships (Fig. 8B), it can be seen that after unloading, F_{active} of the concentric contracting fibers of muscle 1 dramatically drops from 84 to 4 N at a mouth opening of 33.5 mm (and at a mouth opening of 24.5 mm from 97 to 9 N). The active force of the eccentric contracting muscle fibers of muscle 2 increases from 5 to 9 N at a mouth opening of 33.5 mm (and at a mouth opening of 24.5 mm from 5 to 8 N). Clearly, the force-velocity properties of muscle 1 (the closers) are mainly responsible for the steep decline in bite force after unloading in the simulations.

Factors contributing to the magnitude of the impact velocity after unloading

Figure 9 shows the impact velocities of the mandible in simulated unloading experiments of 40, 100, and 200 N. The initial mouth opening is set at 33.5 mm and the travel distances are 2.0, 4.0, 6.0, and 8.0 mm. In the simulations, the initial force of muscle 2 was set to be 5.1 N.

To simulate 'natural' biting through hard and brittle food, the influence of the unloading apparatus was switched off ($F_{resistance} = 0$ N after $t_{unloading}$ and $mass_{lb} = 0$ kg). The histogram in Figure 9A shows the results. As can be seen, the impact velocity does not increase at distances of travel > 4-6 mm (a dynamic equilibrium is reached between the opening and closing forces at that point).

The contribution of the active force-length characteristics of the muscle fibers to the magnitude of the impact velocity was studied by leaving them out of the model (Fig. 9B). Without force-length properties, the impact velocity increases by 31% at a travel distance of 8.0 mm and $F_{start} = 40$ N, by 13% at $F_{start} = 100$ N, and by 7% at $F_{start} = 200$ N.

By leaving the force-velocity characteristics of the muscle fibers out of the model, the impact velocity increases tremendously (Fig. 9C); in the 40 N condition at a mouth opening of 33.5 mm and over a travel distance of 8.0 mm, an increase of 162% is calculated, and in the 100 and 200 N condition, both have an increase of 194%. Differences between the 40, 100, and 200 N simulations mainly are due to the passive muscle components which are not velocity dependent. In the 40 N experiments, the contribution of these passive components

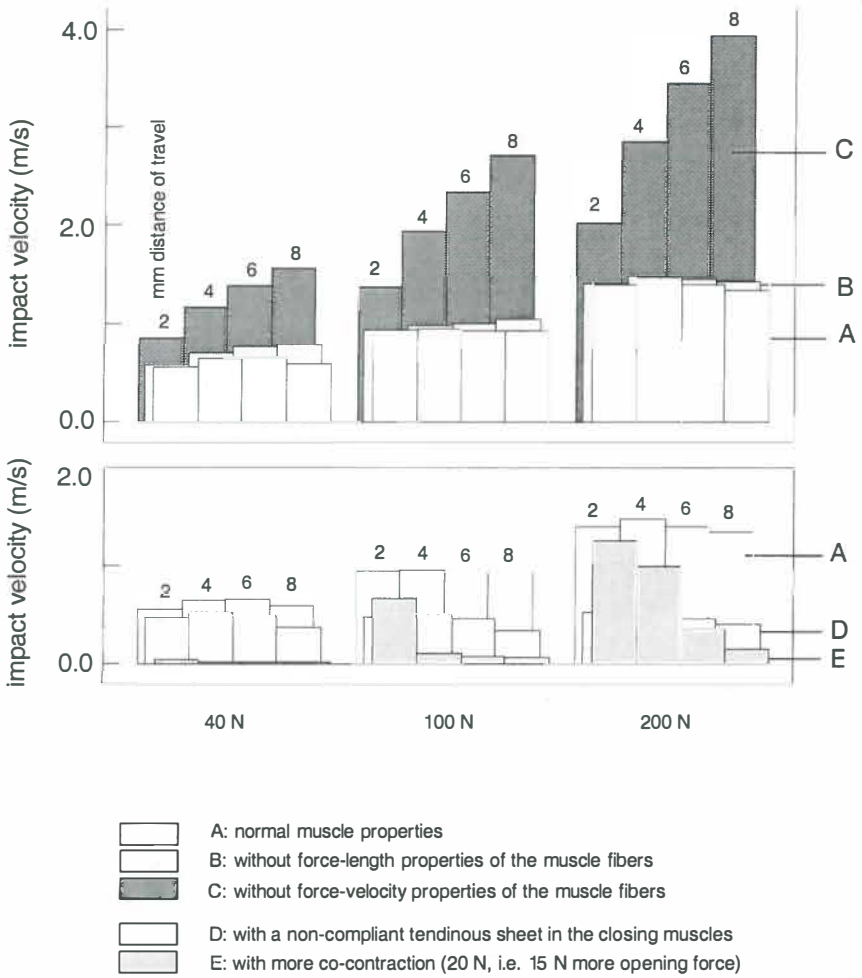


Figure 9. Impact velocities as predicted by the model for an unconstrained human mandible (no unloading device present), at an initial force of 40, 100, and 200 N, an initial mouth opening of 33.5 mm, and distances of travel of 2.0, 4.0, 6.0, and 8.0 mm. The contribution of the various biomechanical factors to the impact velocity was studied by leaving them out of the model.

is relatively large.

The contribution of the compliance of the tendinous sheet of the jaw-closing muscles to the impact velocity was explored by reducing it to zero so that the sheet could not take up length (Fig. 9D). This gave rise to a reduction of impact velocity of 37% (40 N simulations), 63% (100 N simulations), and 70% (200 N simulations) at a distance of travel of 8.0 mm. Apparently, the compliance of a tendinous sheet in the jaw-closing muscles favors the impact velocity.

Finally, the influence of the co-con-

tracting jaw muscles on the impact velocity was explored by increasing $F_{openers}$ from 5.1 to 20 N (Fig. 9E). This increase in co-contraction caused the impact velocity to become 0.03 m/s after a distance of travel of 2.2 mm in the 40 N simulations. In the 100 N and 200 N simulations, the impact velocity levelled out at 0.06 and 0.16 m/s above a travel distance of 4.4 and 7.6 mm, respectively.

Sensitivity analysis

The sensitivity analysis shows a dominant influence of the parameters of the passive force-length curve of the muscle fibers in both muscles, the parameter determining the stiffness of the tendinous sheet of both muscles, the optimal length of the fibers and tendinous sheet of muscle 1, the fiber type composition of muscle 1, the initial force exerted by the co-contracting jaw-opening muscles, and the mass of the lower bar. An increase in parameter value of 1% of any of the above dominant parameters gives a change in the model force output of 0.1-0.9%. The model is robust to changes in the other parameters of the jaw model, giving a change in the model force output of 0.0-0.07% at a 1% increase.

Discussion

In this study, we showed that the model fits the experimental results, as described in chapter 2, well. Not only do the impact velocities match, but more importantly, the time-varying output forces that result in these impact velocities match also in great detail. Figure 6 illustrates that the model predicts somewhat lower impact velocities than measured. This is due to noise in the position signal of the experiments; maximal velocities were calculated from this noisy position signal resulting in slightly higher values of the impact velocities than would be available after curve fitting.

Limitations of the model

In modeling, one is forced to choose a compromise between realism and elegance. When a model is complex and data are far and few between, too many unknown values of parameters make the model easy to match any data set, whereas the underlying processes may not be in agreement with reality. At the opposite end, an elegant model may not be able to simulate the actual background of some observed phenomenon. In the present study we have looked for a compromise. Therefore, we started with a model that was obviously too simple, and added elements until the model was able to simulate the experiments. The good agreement between model and experiment is primarily due to the facts that sufficient subprocesses were modeled (such as intramuscular dynamics) as well as sufficient muscle physiological properties, and that the model contains seven parameters (Table A4A) that could be tuned within sufficiently wide physiological boundaries. To keep the number of free parameters low, we looked for data in literature.

The composition of the model is formed by two dynamic skeletal muscle models connected in an antagonistic way to a representation of the lower jaw, which connects to a model of the experimental device. We

founded the muscle models on the muscle model of Otten (1987a).

Basically these muscle models are a Hill-type model (Hill, 1938), which has force-velocity properties that are not history dependent. This choice may give some unrealistic effects in force production, which may have been covered up by parameter tuning.

To come to anatomical realism, morphometric data from a cadaver study were used in the muscle models. Different action lines of the muscle fiber bundles of a single jaw muscle were simplified to one action line and the diversity in compartmentalization and variation in recruitment of motor units within a single muscle was ignored.

The geometrical transformation of the coordinates of the sites of attachment of the opening muscles resulted in a rather small value of the length of muscle 2. This caused a relative large change in muscle fiber length of the openers at small movements of the jaw. However, the human hyoid bone moves considerably during normal oral behavior (Pancherz et al., 1986; Thexton et al., 1981). Because in the present model approach we did not wish to simulate hyoid movements, we kept the position of the hyoid bone constant. This invokes an overestimation of the muscle fiber velocities of the opening muscles. The hyoid bone had a different position from that found in the morphometric data set (see APPENDIX, Morphometric parameters) to avoid opening muscle insufficiency at large mouth openings.

Recruitment of both muscles was kept constant during the whole simulation, which is based on the observation that before unloading the bite force was relatively constant in the experiments and that after the unloading, reflex events evoked in the jaw muscles are too late to have an effect on the bite force during the dynamic phase (Miles & Wilkinson, 1982; Van Willigen et al., 1997). This is in contrast with studies on perturbations of the limb, in which reflex events can have an influence on the control of the limb movement because the duration of the movement is much longer (Angel et al., 1965; Dufossé et al., 1985; Soechting & Lacquaniti, 1988).

The optimization procedure found a value of 5.1 N for $F_{openers}$. Comparable low levels of co-contraction are experimentally found for the anterior digastric muscles (Miles et al., 1982; Van Willigen et al., 1997) and for the inferior head of the lateral pterygoid muscle (Yoshida & Inoue, 1995). Information on the activation of other opening muscles is unavailable.

Results of the model study

The model simulations suggest that the impact velocity of the mandible is limited by the force-velocity properties of the jaw-closing muscles. Without these force-velocity properties the impact velocity would increase by a factor of 1.94-2.7 and 3.9 m/s at an initial force of 100 and 200 N, a mouth opening of 33.5 mm and a travel distance of 8.0 mm; the kinetic energy at impact would be 8.6 times larger in this situation. We therefore conclude that the force-velocity properties of the jaw-closing muscles are a key factor in preventing damage to the teeth after sudden unloading of the jaw.

The active and passive force-length properties of the muscles hardly play any role in limiting the jaw impact velocity. The

simulations suggest that small differences in impact velocities at different mouth openings (at equal initial forces and travel distances) are to be attributed to the passive force-length properties of the jaw-closing muscles. The relative contribution of these passive forces to the total force is greatest at large mouth openings and small initial forces. These passive forces do not decrease at increasing jaw velocity in contrast with active forces.

The model suggests that co-contraction of the jaw-opening muscles -if sufficiently high- helps in limiting the impact velocity. However, this factor may be of less importance than the simulations suggest, due to the wrong assumption that the position of the hyoid bone is fixed during mouth closure. Thexton et al. (1981) and Pancherz et al. (1986) describe a movement of the hyoid bone in an upward-forward direction during mouth closure, which limits the velocity of the fibers of the opening muscles.

Model simulations with an uncompliant tendinous sheet of the jaw-closing muscle predict a reduction of impact velocity of 37-70% over travel distances of 8.0 mm. Apparently this compliance is unfavorable over these distances. In the simulations the tendinous sheet takes up most of the muscle length change after unloading so that the velocity of the muscle fibers remains low, reducing the loss in active muscle force due to the force-velocity properties of the fibers. One could ponder on the functional meaning of tendinous sheet compliance in human jaw-closing muscles. Although it is unfavorable for the impact velocity, compliance may be an unavoidable design factor when packing many short fibers in a limited space, because long tendinous sheets usually are encountered in this kind of muscle architecture.

Looking at the results in a broader context, our study suggests that in particular, the force-velocity properties of the active agonist muscles offer a powerful mechanism for handling effects of sudden perturbations. This mechanism is quick and independent of neural feedback. We submit that the physiological properties of muscles and tendinous sheets should have a more prominent place in theories on motor control.

TABLE A1. Parameter values from literature used in both muscle models

Parameter	Symbol	Value	Source
Constant in force-velocity relationship	k	0.17	Close 1964 (slow fibers)
of fibers		0.25	Close 1964 (fast fibers)
Maximal shortening velocity of fibers	Vmax	7.1	Close 1964 (slow fibers)
		18.3	Close 1964 (fast fibers)
Roundness of active force-length curve	a	2.46	Otten 1987b; Walker and Schrodt 1974
Skewness of active force-length curve	b	0.545	Otten 1987b; Walker and Schrodt 1974
Width of active force-length curve	s	0.216	Otten 1987b; Walker and Schrodt 1974

Values for Vmax are in fiber length/s.

Acknowledgements
The authors like to thank Dr. P.J.W. Jüch and Dr. M.L. Broekhuijsen for their helpful comments regarding the text of this manuscript.

Program Source
We are willing to submit the computer source of the model described to anyone who applies for it.

Appendix

Parameters, used in the muscle model

LITERATURE-FIXED PARAMETERS

Table A1 gives the literature-fixed parameters, used in both muscle models.

In concurrence with Otten (1987a), we characterized the force-velocity relationship of the muscle fibers by a constant k (determining the curvature of the relationship) and V_{max} (the maximal shortening velocity of the muscle fiber). Both parameters are muscle fiber-type dependent and because the jaw-closing muscles differ in fiber composition from the jaw-opening muscles (Eriksson et al., 1981-1983), we used a fiber-type composition-dependent value of k. This value was found by linearly interpolating between the values k = 0.17 for slow fibers and k = 0.25 for fast fibers (see Table A1). The interpolation was guided by the fiber type composition (see Tuned parameters). The same holds for V_{max}, in which V_{max} = 7.1 fiber lengths/s for slow fibers and V_{max} = 18.3 fiber lengths/s for fast fibers. Both parameters are based on Close (1964). V_{max} is the maximal shortening velocity at full activation. The shortening velocity attainable depends on recruitment (Julian & Moss 1981) and was calculated from V_{max} and recruitment using a relationship from Otten (1987a).

The active force-length relationship of the muscle fibers was described using a formula with three parameters, a, b and s, which determine the roundness, skewness, and width of this curve (Otten 1987a).

Walker & Schrodt (1974) published values for the length of actin and myosin filaments of human muscle fibers. These values were fed into the sarcomere model of Otten (1987b), producing the four line segments of the sliding filament theory (dashed line in Fig. A1). The segments were fitted by the formula mentioned above, producing values for a, b, and s (Table A1), with the constraint that their maxima would coincide (Fig. A1). In this way, we could use a single equation for the force-length relationship and could introduce some Gaussian spread in sarcomere length in parallel muscle fibers in a single muscle. The values obtained were: a = 2.46, b = 0.545 and s = 0.216.

MORPHOMETRIC PARAMETERS

The morphometric parameters used in our simulations were derived from a data set of one of us (Van Eijden). Data were used on sarcomere length, muscle fiber length, and physiological cross-sectional areas as well as three dimensional coordinates of the attachment sides of three jaw-closing muscles [temporalis (anterior and posterior part), masseter (superficial and deep portion), and medial pterygoid muscle], and the four jaw-opening muscles [lateral pterygoid,

TABLE A2. Two-dimensional morphometric data.

Jaw muscles	Cross-section (mm2)	L-sarcomere (μm)	L-fiber Normal	L-fiber (mm)	L-tendon (mm)	L-muscle (mm)
Temporalis ant. p.	770	2.35	0.87	26.04	23.96	50.00
Temporalis post.p.	555	2.30	0.85	24.68	26.92	51.60
Medial pterygoid	600	2.51	0.93	11.11	23.59	34.70
Digastric ant.p.	116	2.75	1.02	21.00	29.10	50.10
Digastric post.p.	116	2.72	1.01	18.70	53.70	72.40
Mylohyoid	212	2.83	1.05	23.30	0.00	23.30
Geniohyoid	97	2.65	0.98	34.10	5.40	39.50
Lateral pterygoid	378	2.78	1.03	16.10	5.70	21.80

digastric (anterior and posterior part), mylohyoid, and geniohyoid muscle]. The morphometric measurements were taken at a mouth opening of ca 3 degrees (equivalent to ca 8.5 mm mouth opening including 4 mm overbite; hereafter 'reference position'). The detailed material and methods are described elsewhere (Van Eijden et al., 1995).

We dimensioned the modeled muscles by taking into account the geometrical arrangement of the actual jaw muscles. For this, we transformed the data of the human cadaver study of Van Eijden. This was done in the following way:

1) A value for tendon length was obtained by subtracting muscle fiber length from muscle length. (We neglected the angle of pennation of the fiber bundles to the aponeuroses, introducing an error of 3.4% at an estimated angle of pennation of 15 degrees.)
2) We projected the three-dimensional points of attachment of the muscles onto the sagittal plane (Table A2). (By doing so, we introduced an error of 4.4% in element length of the jaw-closing muscles and about 13.8% in the jaw-opening muscles).
3) The resultant maximal bite force (Fb_{max}) at the level of the canines was derived from

$$2 \cdot \Sigma F_i \cdot d_i / d_b = Fb_{max}$$

where F_i = maximal force of muscle i ; d_i = length of the moment arm of the muscle vector of muscle i; d_b = length of the moment arm of the bite force vector. This was done separately for the closing muscles and the opening muscles (resulting in $Fb1_{max}$ and $Fb2_{max}$, respectively). [Maximal forces of the jaw muscles were determined on the basis of their cross-sectional area; we used a value of 0.35 N/mm² (Nygaard et al., 1983; Weijs & Hillen, 1985)]. The moment arms are the perpendicular distances between the two-dimensional force vectors and the axis of rotation of the mandibular condyles.

Because the condyles translate 16 mm

TABLE3. Parameters based on morphometric data of a cadaver study

Parameter	Symbol	Muscle 1	Muscle 2
Fb-max bite point	Fbmax	622 N	288 N
effective optimal length tendon	Lo tendon	76.2 mm	29.9 mm
effective optimal length fibers	Lo fibers	75.6 mm	45.1 mm
internal mass in muscle	mi	0.020 kg	0.005 kg

Study was done at a mouth opening of 8.5 mm

anteriorly and 4 mm caudally at a jaw rotation of 30 degrees (Falkenström, 1993; Merlini & Palla, 1988; Obwegeser et al., 1987), the average position of the axis of rotation was determined to be situated 31.6 mm below and 1.9 mm anterior to the center of the condyles. The length of d_b was calculated to be 79.2 mm. $Fb1_{max}$ appeared to be 622 N, whereas $Fb2_{max}$ amounted to 288 N (Table A3).

4) We corrected for the geometrical arrangement of the jaw muscles by multiplying the length of the muscles, fibers and tendons by d_b/d_i. (This magnifies these lengths by a factor of *ca* 3 for the jaw-closing muscles and a factor of *ca* 2 for the jaw-opening muscles.)
5) The jaw-closing and -opening muscles were grouped by taking the weighted average of their muscle, fiber, and tendon length per group. The weight factors used were the maximal force contribution of each muscle after correcting for the force transmission d_i/d_b.
6) From the sarcomere length of each jaw muscle at the jaw reference position, we took a rough average (up to the second decimal) resulting in 2.40 μm for muscle 1 and 2.75 μm for muscle 2. Because the human optimal sarcomere length was found to be 2.7 μm (Fig. A1), we could establish the normalized length of muscles 1 and 2 (0.89 and 1.02, respectively) at the reference position. From this we established that the optimal fiber length of muscle 1 is 75.6 mm and of muscle 2 is 45.1 mm (Table A3).

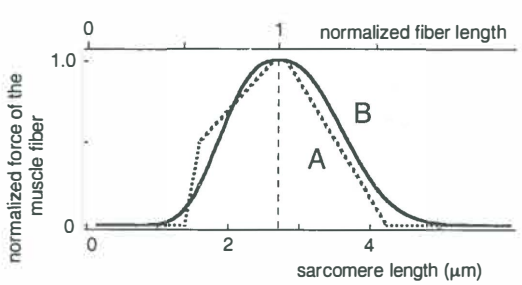


Figure A1. Calculated active force-length curve for human sarcomeres. Dashed line, curve produced by means of the sarcomere model of Otten (1987b) with data from Walker and Schrodt (1974) as input. Solid line, curve produced by a formula suggested by Otten (1987a) for the active force-length relationship. Curve B was fitted to curve A with coinciding maxima.

7) In order to be able to simulate the covered range of mouth opening of 24.5-33.5 mm, we moved the hyoid-bone 5 mm dorsocaudally (without changing the moment arms of the opening muscles) as compared to the position of the hyoid in the material of Van Eijden. This choice was based on Pancherz et al. (1986), who showed a similar hyoid movement when the mouth is opened over a distance of 19 mm. After the above geometric transformation we arrived at a normalized length of muscle 1 of 1.22 and 1.10 and of muscle 2 of 0.71 and 0.91 at a mouth opening of 33.5 and 24.5 mm, respectively (see Fig. 8).

The effective mass of the mandible and the jaw muscles at the level of the incisors was calculated to be 0.230 kg. By effective we mean that we are looking for a mass situated at the incisors that mechanically has the same effect on the dynamics of the motion as the distributed mass of the mandible and jaw muscles. First we calculated the inertia of the mandible and jaw muscles about the axis of rotation in our study from the moment of inertia of the lower jaw and jaw muscles as measured by Koolstra & Van Eijden (1995). When properly chosen, a mass situated at the incisors multiplied by the square distance to the axis of rotation of the lower jaw produces the same inertia as the one calculated.

The values for the internal masses of muscles 1 and 2 in the model were calculated using the total volumes of their muscle fibers and their distances to the center of rotation of the lower jaw (we were interested in the effective masses of the muscles at the bite point). To obtain the volumes, we multiplied the physiological cross-sectional area with the fiber length of each muscle. The masses of the jaw-closing and -opening muscles were calculated to be 0.060 and 0.015 kg, respectively. We divided the masses of the muscles in three equal parts of which one part was linked to the lower jaw, one part included in the model as internal muscle masses (Table A3), and one part was assigned to the unmoving skull or hyoid bone.

Tuned parameters

To obtain full definition of the model, a choice still had to be made of another seven parameter values. These parameters describe the passive force-length properties of the muscle fibers and tendinous sheets, the strain of the tendinous sheets at maximal force, the fiber type composition of the muscles and the force production of muscle 2 (Table A4A).

The values of these parameters were calculated by the optimization procedure while meeting two criteria: the model responses should match as close as possible with the experimental results of Nagashima et al. (1997), and the values of the parameters should be within the physiological range as far as this is known. To establish the parameter values we used the search procedure described by Nelder & Mead (1965). This procedure finds minima of multidimensional functions of which partial derivatives are hard to calculate. Minimizing such functions is impossible with classical steepest descent methods (Press et al., 1986). (Table A4A give the results of this search.)

Expressions for the passive force-length relationships of both the muscle fibers and

TABLE A4. Parameters of the muscle models

Parameter	Symbol	Value
A. Tuned values		
Coefficients of passive force-length relationship	c1-fiber	-14.42
of the muscle fibers according to Otten 1987a	c2-fiber	8.99
Coefficient of passive force-length relationship of	c2-tendon	63.95
the tendons according to Otten 1987a		
Strain tendon at maximal force, %	-	5
Percentage of slow fibers of muscle 1	ft1	88
Percentage of slow fibers of muscle 2	ft2	17
Initial force, produced by muscle 2, N	F-openers	5.1
B. Chosen values		
Passive damping coefficient tendons, Ns/m	b	5

tendinous sheets were used as suggested by Otten (1987a).

Identical passive force-length relationships of the muscle fibers were tuned for muscles 1 and 2 after normalizing for both optimal length and maximal isometric active muscle force. After tuning, they gave a passive force of the jaw-closing muscles of about 20 N at a mouth opening of 33.5 mm, which, by coincidence, appeared to be in line with the results of Miles et al. (1986).

Tendinous sheet strain was kept within boundaries of 4-8% while optimizing and was left by the procedure at a value of 5% of their rest length at maximal isometric muscle force. Experimentally, aponeurotic sheet strain is between 6% and 8% of its rest length (Lieber et al., 1991; Trestik & Lieber, 1993). This still leaves us with a choice for the stiffness of the tendinous sheets. This value was left free in the fitting process but were kept identical for the sheets of muscles 1 and 2 relative to the maximal forces of the muscles.

In the optimized configuration the fiber type composition appeared to be 88% type 1 and 12% type 2 for muscle 1 (after applying boundaries of 0-30% type 2) , and 17% type 1 and 83% type 2 for muscle 2 (after applying boundaries of 60-90% type 2). The applied boundaries are those described by Eriksson et al. (1981-1983).

As a result of the optimization procedure, the force exerted by the co-contracting jaw-opening muscles appeared to be 5.1 N.

Damping properties were added to the tendinous sheets, because hysteresis is a well known property of tendinous material and because they were convenient and necessary in avoiding intramuscular oscillations in the simulations. We chose a low damping coefficient of the tendinous sheets of 5 N s/m (Table A4B).

Device parameters

The contribution of each of the four forces (F_{offset}, F_{magnet}, $F_{vibration}$ and F_{end}) to $F_{resistance}$, and the effective mass of the lower bar at the bite point was established by the above-mentioned search method in which the behavior of the device model was matched with that of the unloading apparatus. The effective mass of the lower bar at the bite point was 1.40 kg.

4

The riddle of the large loss in bite force after fast jaw-closing movements

Summary

In unloading experiments (in which experimentally the resistance to a forceful static bite is suddenly removed), it is shown that the residual bite force (when the jaw system is arrested shortly after the unloading) is remarkably small. For example, of a 100 N initial bite force only 18 N is left after a distance of jaw travel of 5.0 mm. The present experiments are designed to study whether the magnitude of the low residual bite force is dependent on the initial bite force, the initial degree of mouth opening and the distance of jaw travel. Furthermore, it is analysed whether the low magnitude of the residual force can be attributed to reflex events of the jaw muscles or to the force-length properties of the jaw-closing muscles. It is shown that the residual forces are largely dependent on the distance of jaw travel and are barely sensitive to variations in initial mouth opening. The relative residual forces are independent of the magnitude of the initial bite force. The maximum in the residual forces is in the order of 25% of the initial bite force after a jaw travel of 4.5 mm. The low values of the residual forces can not be attributed to reflex events, because it takes about 80 ms for the masseter muscles to decrease their force to a 50% level after their excitation is switched off. Furthermore, it is shown that the force-length properties of the jaw-closing muscles are not responsible for the small values of the residual forces, since over the trajectories used in the present experiments, the sarcomere lengths of the jaw-closing muscles are beyond their optimum sarcomere length. It is suggested that the low residual forces are brought about by (1) a non-uniform sarcomere behaviour of the jaw-closing muscles when contracting or (2) a long lasting change in the myofilament system of the closing muscles induced by the sudden shortening of muscle fibers.

Introduction

In unloading experiments (in which the resistance to a forceful static bite is suddenly removed) it is shown that bite forces decrease quickly as soon as the mouth starts closing (Hannam et al., 1968; Miles & Wilkinson, 1982; Van Willigen et al., 1997). For example, of an initial bite force of 100 N, the bite force is reduced within 14 ms to about 5 N after a distance of travel of 5.0 mm, while the jaw is still moving (Van Willigen et al., 1997). This quick decrease in force can largely be explained by the force-velocity properties of the jaw-closing muscles (Slager et al., 1995; Nagashima et al., 1997): they lose a fair amount of their force when they shorten, resulting in vanishing of the bite force.

However, also when the mandible is arrested shortly after the unloading, the residual bite force (i.e. the force when the jaw system is in a static condition again after the impact) appears to be remarkably small (Van Willigen et al., 1997). For example, of an 100 N initial bite force only 18 N is left when the mandible is stopped after a distance of travel of 5.0 mm.

This low magnitude of the residual force can hardly be attributed to the force-velocity properties of the jaw-closing muscles, since these muscles have finished their shortening process after the mandible has come to a standstill.

Also co-contraction of the jaw-opening muscles as a major source of the small magnitude of the residual force is improbable, since co-contraction is weak (Miles and Wilkinson, 1982; Van Willigen et al., 1997), and Van Willigen et al. (1997) were unable to detect any relationship between the magnitude of the residual force and the amount of co-contraction.

With regard to reflex events (Hannam et al., 1968; Lamarre & Lund, 1975; Miles & Wilkinson, 1982; Yoshida & Inoue, 1995; Van Willigen et al., 1997), there is a silent period in the EMG of the jaw-closing muscles (delay 5-20 ms) and a reflex-activation of the jaw-opening muscles (delay 20-40 ms) after the onset of movement of the mandible. However, due to the reflex latencies and the electro-mechanical delay of the jaw muscles, the influence of these reflex events on the bite force is also probably small. The present study concentrates further on possible explanations of the low residual forces mentioned.

Unloading experiments were performed to determine whether the magnitude of the residual bite force is dependent on the initial bite force, the initial degree of mouth opening and the distance of jaw travel. This was done by asking subjects to bite on a pair of rigid bars of which the lower bar could be suddenly released. After a certain distance of travel the lower bar was arrested. The movement of the lower bar and the forces exerted on it were recorded. The magnitude of the residual force was determined under circumstances of four initial bite forces, four initial degrees of mouth opening and three distances of jaw travel. This gave 48 different experimental conditions.

A possible influence of the force-length properties of the jaw-closing muscles on the magnitude of the residual force was analysed in a model study. For that, data of Van Eijden et al., (1992, 1995 and 1996) were

used on sarcomere lengths of human jaw-closing muscles at various mouth openings. These data were transformed to sarcomere lengths matching the trajectories of jaw movement used in our unloading experiments. Thereafter, the isometric active forces were calculated in the range of interest.

Finally, possible influences of reflex events of the jaw muscles on the residual force were analysed. Since there is no information available on the time course of the decay in force of human jaw muscles when their excitation is switched off, the electro-mechanical delay of masseter muscles was measured. This was done by direct electrical stimulation of both masseter muscles.

Material and methods

Participants

Five dentate individuals participated in the experiments. All were free from signs of muscular or temporomandibular dysfunction. The participants were fully informed about the experimental conditions. They gave their informed consent and participated in a protocol that complied with Dutch law.

Throughout the experiments the participants sat in front of an unloading device as shown in Figure 1. The position of the device was adjusted in height so that it was at the level of the mouth. Bite forces were exerted between the upper and lower incisors and cuspids.

Unloading device

The device contained two parallel aluminum biting bars (80 mm long, 50 mm wide and 6 mm thick). The upper bar was attached to two vertical plates mounted on a ground plate. The lower bar either hinged around an axis (unloading mode) or was locked by inserting an additional pin (force-transducer mode). The lower bar was fixed to the hinging axis on which a magnet was mounted so that the angular displacement of the lower bar could be recorded by a magneto-resistive-sensor (Philips KM11OBH/21). From the angle of rotation, the displacement of the bite area of the lower bar was calculated. For the subject's comfort, the bite areas of the bars were covered with a layer of sticking plaster.

Two pairs of balanced strain gauges were attached on either side of the lower bar, 25 mm from the mouth piece, measuring the bite force applied to the lower bar independently of the point of impact (Stegenga, 1991).

In the unloading mode the initial resistance to closing was achieved by an empowered solenoid of which the core was fixed to the lower bar by means of a steel cable; the solenoid itself was fixed to the body of the device. The movement of the lower bar was thus prevented by the core of the solenoid, being held in place by the magnetic field of the solenoid. The magnetic field of the solenoid could be switched off (triggered by the output of the strain gauges) at a voltage equivalent to a force of 100, 80, 60 and 40 N. When the solenoid was switched off, the (bite) force on the bitten end of the lower bar suddenly lacked its counter force and accelerated the bar; as a result the lower bar dropped at the back and its front was lifted up.

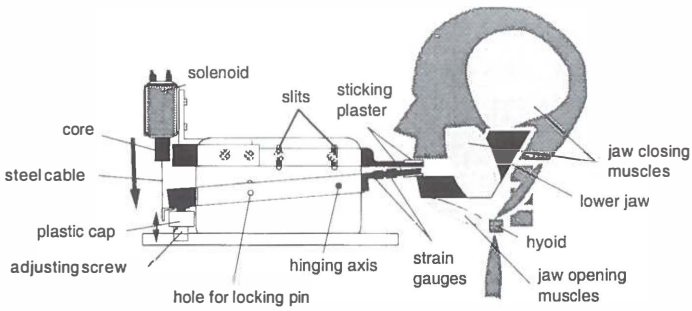


Figure 1. 'Unloading' device and force-transducer combination: two parallel aluminum biting bars (the lower bar with strain gauges) are attached to two plates mounted on a heavy support; the lower bar is fixed to an hinging axis of which the angular displacement is measured; the initial resistance to closing is achieved by a solenoid.

The position of the upper bar could be varied by means of slits and bolts so that the initial mouth opening of the subjects at the start of each experiment could be adjusted between 24.5 and 33.5 mm when biting on the device. The mouth opening of the subjects was defined as the inter-incisor distance added to the vertical overlap of the dentition (overbite).

The distance to travel of the bitten end of the lower bar could be modified by means of an adjusting screw which was situated in the ground plate near the end of the lower bar (Fig. 1). To buffer the shock of collision the head of the screw was covered with a soft plastic cap.

The mechanical properties of the unloading apparatus were established in a series of tests described in previous studies (Van Willigen et al., 1997; Nagashima et al., 1997).

Protocol

With the apparatus in the unloading mode, subjects were asked to bite carefully through a resistance of 100, 80, 60 or 40 N; this was done with an initial mouth opening of 33.5, 30.5, 27.5 or 24.5 mm and a distance of travel of the mandible of 1.5, 3.0 or 4.5 mm after unloading. This gave 48 experimental conditions. All experiments were repeated 5 times giving a total of 240 observations per subject.

Data analysis

For each experimental condition the results were pooled and averaged. Figure 2 depicts such an average of the force (upper trace) and position recordings (the bottom trace) of a 100 N unloading experiment with an initial mouth opening of 24.5 mm and a distance of travel of 4.5 mm (n=25, standard deviation in grey). The middle trace shows the calculated average velocity, i.e. the velocity of the lower bar/lower teeth combination calculated from the position recordings of the bitten end of the lower bar. This was done by differentiation of the position signals over time.

The moment of unloading was defined as the moment that the bite force dropped (Fig. 2, top panel, point 1).

The time of impact of the lower bar/mandible combination was the moment at which the velocity reached its maximum (point 2 in the velocity display of Fig. 2). This point coincides with the first moment of contact of the back of the lower bar with the plastic cap covering the adjusting screw; after this instant the velocity dropped to zero.

The time span between the unloading and the moment of impact was called 'the dynamic phase' of the experiment (Fig. 2 in grey), and the period after the impact 'the post-dynamic phase' (Fig. 2 in white). Forces measured in the latter period were called 'the residual forces. To quantify these residual forces we determined the first maximum in the force signals 15 ms after the moment of impact (dark circle in top panel of Fig. 2). The boundary of 15 ms was taken to exclude transient force peaks caused by the collision of the back of the lower bar with the head of the adjusting screw after impact (point 3 in the top panel of Fig. 2).

Electro-mechanical delay of the masseter

For the interpretation of our results, we measured the electro-mechanical delay of the masseter muscles in an additional experiment. For that the lower bar of the device was locked so that it could be used as a simple force transduce. We asked the subject to make tooth contact with the upper

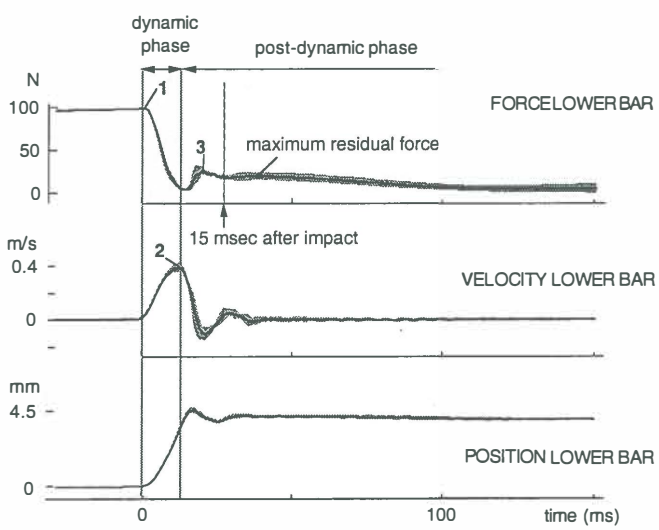


Figure 2. Outcome of an unloading of a 100 N static bite commencing at mouth opening of 24.5 mm and with a sudden arrest of the jaw after a traveled distance of 4.5 mm. The panels show the average and the standard deviation (in grey) of the lower bar bite force signal, the (calculated) velocity of the lower bar and the position of the lower bar relative to its starting position. (1) Time of unloading and magnitude of the initial bite force at unloading; (2) time of impact of the lower bar and the value of its maximal velocity; (3) force transient evoked by the impact. The maximum of the residual force is indicated by a dark circle.

and lower bar (interincisor distance 24.0 mm) and relax. Thereafter the left and right masseter were stimulated (direct electrical transcutaneous stimulation) with two bipolar electrodes (diameter 22 mm, electrode-distance 5 mm). The stimulus took the form of a 50 Hz pulse constant current (pulse duration: 1 ms, 2.5 Hz amplitude modulation) provided by a Phyaction 787 S/N 21252 stimulator, used in physiotherapy. Stimulus strength was increased until the resultant bite force reached a plateau of about 20 N for 3 seconds (maximal stimulus about 25 mA). Then the stimulus was switched off. Both the bite force and the stimulus were recorded. The experiment was repeated 5 times.

The half-relaxation time of both masseter muscles was defined as the time elapsed from the first decay in bite force until the force reached its 50% level (Fig. 3).

Force-length considerations

The relationship between the force-length properties of the active jaw-closing muscles and the magnitude of the residual bite forces measured was analysed in a model study. This was done in the following way.

First, an active force-length curve of a human sarcomere was calculated. This was done by feeding the data of Walker and Schrodt (1974) -on the length of actin and myosin filaments of human muscle fibers- into the sarcomere model of Otten (1987b). This gave four line segments (two ascending parts, a plateau, and a descending part) describing the force-length relationship of a human sarcomere in general (solid curves in Fig. 4).

However, in reality, force-length curves of muscle fibers are not so angular, since sarcomeres vary in length between muscle fibers. Therefore, to get a more realistic force-length curve, the force-length curve calculated was smoothed. This was done by fitting the four line segments by a formula describing the active force-length relationship of muscle fibers (Otten 1987a) with the constraint that their maxima should coincide. This resulted in the bell-shaped curves (with an optimum at 2.70 μm) as drawn in Figure 4 (interrupted lines). These curves were used in our analysis.

Based on cadaver studies, Van Eijden et al. (1992, 1995 and 1996) give sarcomere lengths of the anterior and posterior parts of the temporalis, masseter and medial pterygoid muscle, measured at 4 degrees rotation of the mandible. To get an impression of the length of sarcomeres at various mouth openings, the data of Van Eijden et al. were transformed to mouth openings between 0 - 30 degrees. This was done by using a centre of rotation halfway the ramus of the mandible (Van Eijden et al., 1992 and 1995) or by using a model, simulating a normal jaw-opening movement (Van Eijden et al., 1996). In the present study these transformed data were used to determine the sarcomere lengths (in μm) of the muscles mentioned above at trajectories of jaw movement used in our experiments [i.e. 33.5 mm to 29.0 mm, and 24.5 mm to 20.0 mm; interincisal distance added to the overbite]. This was done by multiplying data on mouth openings (in degrees) of Van Eijden et al. (1992, 1995 and 1996) by a factor of 1.5 (Dijkstra et al., 1995) giving mouth openings in mm. The ranges in sar-

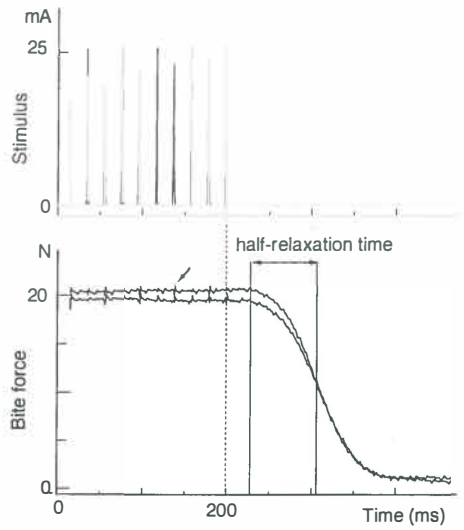


Figure 3. Two superimposed recordings of the resultant force output of both masseter muscles when directly stimulated electrically (bottom panel) and the stimulus applied (top panel). Stimulus artefacts can be seen on the bite signal (arrow). At time t=200 ms the stimulus was switched off. The half-relaxation time of the masseter is about 80 ms.

comere lengths matching the experimental trajectories for the temporalis, masseter and pterygoid muscle were drawn into the calculated isometric force-length-curves of the sarcomeres (Fig. 4, anterior parts of the jaw-closing muscles in dark-grey; posterior parts in light-grey).

All processing was done on an Apple Macintosh IIx computer system extended with a Rocket 33 accelerator board (Radius) and running 'DataMonster 2.0', an analysis, graphics and simulation program written by one of the authors (E.O.)

Results

The experiments

Figure 5 depicts a selection of the averaged force recordings (n=25, standard deviations in grey) as measured in the unloading experiments. Figure 6 shows the averages of the maximum residual forces (relative to the initial bite force =100%) and their standard deviations (n=25). It can be seen that in all experimental conditions there was a considerable loss in bite force in the dynamic phase after the unloading (Fig. 5), and there were small residual forces in the post-dynamic phase (Fig. 5 and 6).

The results of the experiments were remarkably reproducible as can be seen from the small standard deviations (SD). Moreover, the variation in force profiles at various degrees of mouth opening (MO) was quite small (compare Fig. 5A with Fig. 5B).

The magnitude of the residual forces appeared to be dependent on the initial bite force (Fig. 5A-B). However, the time courses and relative magnitudes of the residual forces were independent of the magnitude of the initial bite force (Fig. 5C and Fig. 6). This is clearly illustrated in Figure 5C, which shows superimposed average relative force recordings at four different initial bite forces (distances of jaw travel (TD) 1.5 and 4.5 mm; MO 24.5 mm).

The magnitude of the (relative) maxima in the residual force was mainly dependent on the distance of jaw travel (Fig. 5D): large travel distances gave small residual forces in an unloaded jaw system. The average relative maximum residual forces (Fig. 6) were between 58 to 63% (SD < 7%) of the

initial bite force at a TD of 1.5 mm, 33 to 43% (SD < 6%) at a TD of 3.0 mm, and 18 to 31% (SD < 7%) for a TD of 4.5 mm.

The loss in bite force after unloading was barely sensitive to variations in initial mouth opening. This is clearly illustrated in Figure 5E (initial bite force 100 N; TD 4.5 mm). Variations in force in the dynamic phase were negligible, whereas the residual forces and their maxima (dark circles in Fig. 5E) only showed small differences. The maximum residual force was 27 N (SD=5 N, n=25) when the unloading started at a MO of 33.5 mm, and 19 N (SD=5 N, n=25) at a MO of 24.5 mm.

The time of occurrence of the maxima in the residual forces (dark circles in Fig. 5) was between 34 and 44 ms after the unloading.

The electro-mechanical delay of the masseter

Figure 3 shows (bottom panel) two superimposed recordings of a bite force evoked by direct electrical stimulation of both masseter muscles. The top panel shows the stimulus. The bottom panel shows the bite force (note stimulus artefacts, picked up by the lower bar strain gauges). At time t=200 ms the stimulus was switched off. The half-relaxation time of the masseter appeared to be about 80 ms (Fig. 3). The delay from the last electric pulse until the first decrease in force was about 25 ms.

Influence of force-length properties on changes in isometric active force

Figure 4 displays for the jaw-closing muscles the changes in sarcomere length within the trajectories of jaw movement used in our experiments. The dark circles indicate the (established) length of the sarcomeres (and the corresponding isometric force) at the start of the unloading, whereas the open circles are sarcomere lengths when the lower bar/lower teeth combination has reached its end position after a TD of 4.5 mm (Fig. 4A, MO 33.5 mm; Fig. 4B, MO 24.5 mm). As can be seen, changes in sarcomere length and changes in the corresponding isometric force were relatively small. Moreover, at unloadings at a MO of 33.5 mm (Fig. 4A) the sarcomeres appeared well beyond their optimum length (except for the posterior part of the masseter muscle), which leads to an increase in isometric force of about 15% of the maximal isometric force after a TD of 4.5 mm.

At a MO of 24.5 mm (Fig. 4B) the sarcomeres of the posterior parts of the temporalis and masseter muscle were below their optimum length, resulting in a -small- decrease in isometric muscle force at jaw closure; the sarcomeres of the anterior parts of the temporalis and masseter muscle, and of the medial pterygoid muscle were beyond their optimum length so that they increased their force at jaw closure.

Discussion

In line with Van Willigen et al. (1997), the present study shows that residual forces -as measured when the jaw is arrested shortly after the unloading- are remarkably small; this holds even for experiments with a distance of jaw travel of only 1.5 mm.

Although the magnitude of the residual

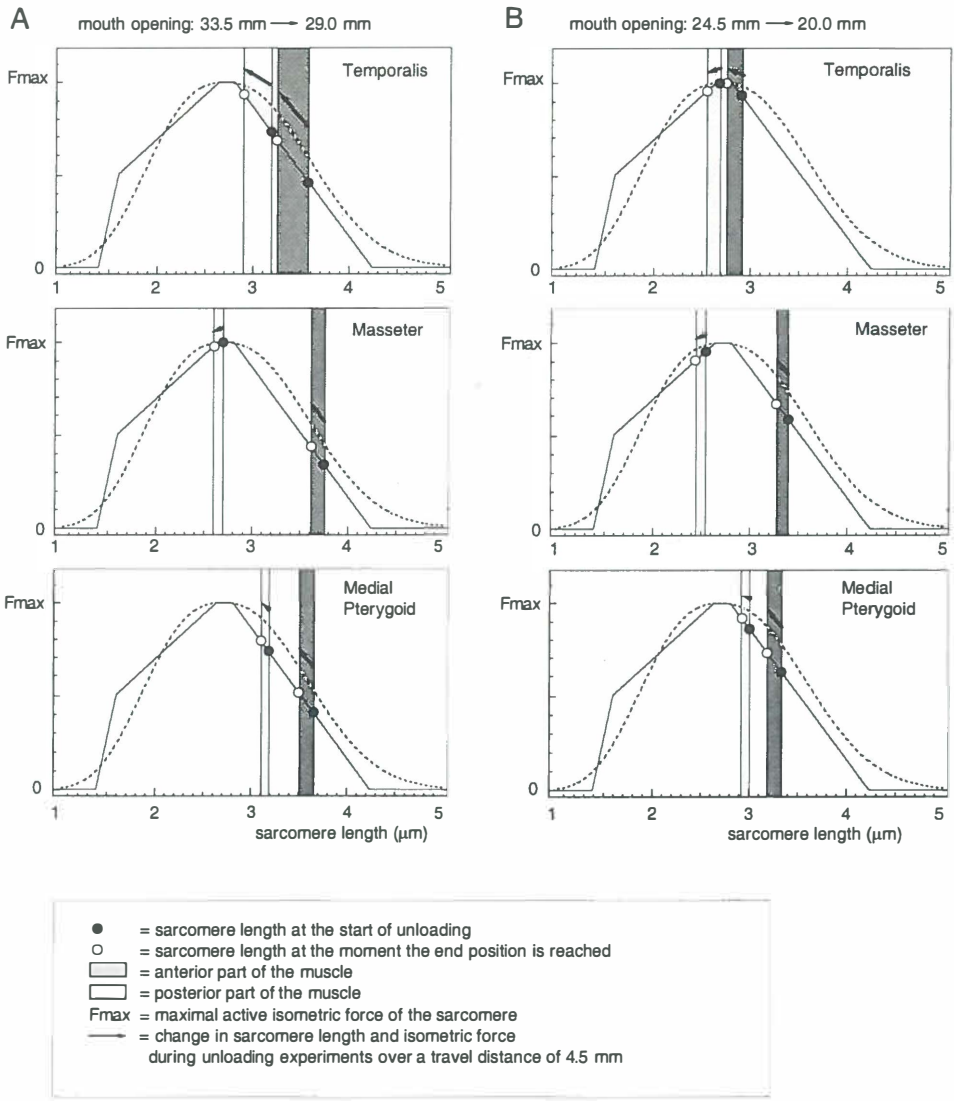


Figure 4. All panels display identical model simulations of an isometric force-length-curve of a human sarcomere (solid curves, model outcome; dashed curves, smoothed active force-length curve). In the separate panels changes in sarcomere length within the trajectories of jaw movement used in our experiments are drawn for the temporalis, the masseter and the pterygoid muscle. (A) Experiments commencing with a mouth opening of 33.5 mm. (B) Unloadings starting at a 24.5 mm mouth opening. Note that sarcomere lengths of the jaw-closing muscles are *beyond* their optimum, resulting in an *increase* in isometric muscle force at jaw closure.

force is dependent on the initial bite force, the relative maximum residual forces are independent of the strength of the initial bite. Furthermore, the magnitude of the maximal residual forces appears to be largely dependent on the distance of jaw travel and barely sensitive to variations in initial mouth opening.

There are five factors which possibly underlay the observed phenomena: (1) reflex events, (2) co-contraction of the active muscles, (3) active force-length properties of the jaw muscles, (4) active force-velocity properties of the jaw muscles together with tendon creep and (5) structural properties of the myofilament system of the jaw muscles. In the following paragraphs these factors will be discussed.

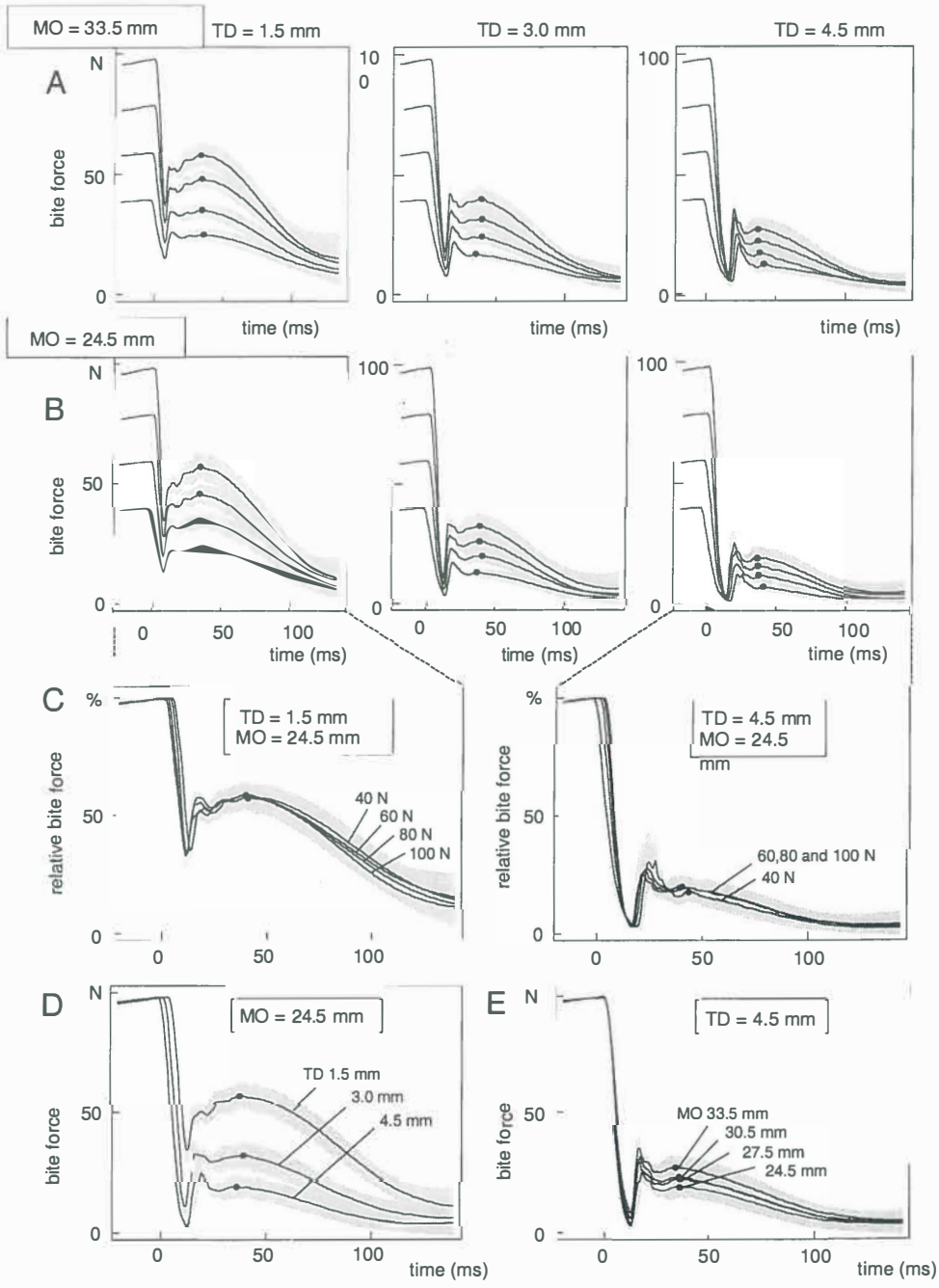
Reflex events

Depending on the experimental condition, the maxima of the residual forces occurred between 34 and 44 ms after unloading, whereas there is a silent period in the masseter muscles (delay 9-20 ms after unloading) and a reflex burst (delay 5-80 ms) in the digastrics (Van Willigen et al., 1997). In other words, the maxima of the residual forces occurred after these reflex events. On first sight this explains the reduction in bite force after the unloading.

However, we measured a half-relaxation time in masseter muscle force of about 80 ms. This implies that the masseter muscles only have lost a fraction of their force due to the onset of the silent period when the residual force is at its maximum. This is illustrated in Figure 7 which shows an average force recording (of an unloading experiment of 100 N with a travelled distance of 4.5 mm; curve A), and the decline in isometric force of the masseter muscles due to a silent period with a delay of 9 ms after the unloading (curve B). For reasons of clarity the output trace of the masseters (curve B) is scaled to 100 N. As can be seen, the maximum residual force (point 1 on curve A) is reached at a moment that the declining force of the jaw-closing muscles still has a level of 90% of its initial value (point 2 on curve B).

A similar discussion can be made for the reflex burst in the jaw-opening muscles. Therefore, due to their electro-mechanical delays, the silent period in the jaw closers and the reflexes in the jaw-openers, both

Figure 5. (A and B) Results of unloading experiments starting with initial bite forces of 40, 60, 80 and 100 N, traveled distances (TD) of 1.5, 3.0 and 4.5 mm, and initial mouth openings (MO) of 33.5 mm (A) and 24.5 mm (B). The panels depict average forces (n=25; SD, grey areas). Dark circles, maxima in residual forces. Note that the residual force is dependent on the initial bite force. (C) Comparison of average residual forces relative to the initial bite force (=100%) of 40, 60, 80 and 100 N unloading experiments (TD 1.5 and 4.5 mm; MO 24.5 mm). Note that the relative residual force is independent of the initial bite force. (D) Comparison of average residual forces (100 N) after three different distances of jaw travel (TD 1.5 mm, 3.0 mm and 4.5 mm). Note that the residual force is mainly dependent on the distance of jaw travel. (E) Comparison of average residual forces of four 100 N unloading experiments starting at mouth openings of 33.5, 30.5, 27.5 and 24.5 mm and the same traveled distance of 4.5 mm. Note that the residual force is barely sensitive to the initial mouth opening.


MO = 33.5 mm
TD = 1.5 mm
TD = 3.0 mm
TD = 4.5 mm
A
N
bite force
50
0
10
0
100
0
time (ms)
MO = 24.5 mm
B
C
%
relative bite force
TD = 1.5 mm
MO = 24.5 mm
40 N
60 N
80 N
100 N
TD = 4.5 mm
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E
TD = 4.5 mm
MO 33.5 mm
30.5 mm
27.5 mm
24.5 mm

being part of the motor control of the lower jaw, are inadequate in reducing the bite force to the observed levels.

Co-contraction

It seems unlikely that co-contraction of the jaw-opening muscles is very helpful in reducing the bite force.

First, there is hardly any correlation between the amount of co-contraction of the digastric muscles and the loss in bite force in the post-dynamic phase (Van Willigen et al., 1997).

Second, the inter- and intra-individual variation in co-contraction of the digastric muscles is considerable (Van Willigen et al., 1993 and 1997), whereas this variation in the residual forces is surprisingly small as can be seen from the small standard deviations of the pooled force profiles (Fig. 6).

Third, the levels of co-activation of the anterior digastric muscles (Miles & Wilkinson, 1982; Van Willigen et al., 1997) and of the inferior head of the lateral pterygoid muscles (Yoshida et al., 1995) are low. For example, for the digastrics, the maximal level of co-contraction of the digastric muscles is in the order of 25% of their maximal voluntary contraction in 100 N unloading experiments (Van Willigen et al., 1997).

Sharkey et al. (1984) found an average maximal opening force of about 120 N, generated by all opening muscles together. If we assume that all jaw-opening muscles co-contract at a level of the digastrics, this would suggest a force in the opening direction of ca 30 N in our experiments. This implies that, if the jaw-opening muscles were responsible for the small residual forces measured, they would have increased their force by a factor of 3.5-4 over distances of travel of 4.5 mm, while the length of their muscle fibers only increased by 9%. This is highly implausible. [The latter statement is based on the observation of T.M.G.J. Van Eijden (personal communication) that the fiber length of the jaw-opening muscles is about 25 mm, resulting in a lengthening of the jaw-opening muscles of 2.25 mm at travel distances of 4.5 mm of the front teeth.]

Finally, possible effects of co-contraction of the jaw-opening muscles might be counteracted by movements of the hyoid bone which moves in parallel with the mandible during closure (Thexton et al., 1981; Pancherz et al., 1986). If the hyoid moves also parallel with the mandible during unloading, changes in length of the opening muscles will be limited, so that the contribution of the co-contracting jaw openers to the reduction in bite force will be very small.

Force-length properties

When the jaw makes a closing movement (as in our unloading experiments), the length of the sarcomeres of the jaw-closing muscles decreases (Fig. 4), whereas the sarcomere length of the jaw-opening muscles increases. Since the isometric active force that a muscle fiber can produce depends on the length of its sarcomeres, changes in muscle length introduce changes in the isometric forces that an active muscle can exert.

Our model study gives evidence that at trajectories of jaw closure used in our experiments, sarcomere lengths of the jaw-closing muscles are beyond their optimum sarcomere length (i.e. on the descending part

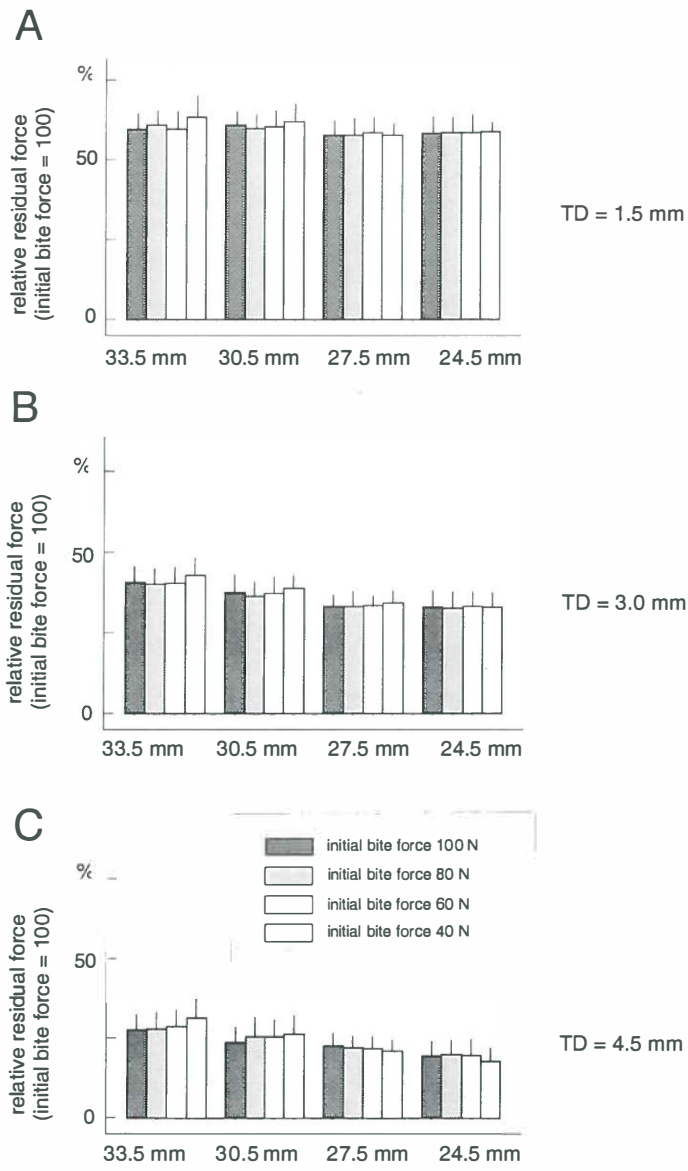


Figure 6. Relationship between the maximum of the residual force (relative to the initial bite force=100%), the initial bite force (100, 80, 60 and 40 N), the initial mouth opening (33.5, 30.5, 27.5 and 24.5 mm) and the travel distances of the mandible (TD): (A) 1.5 mm, (B) 3.0 mm and (C) 4.5 mm. Note that the maxima in the residual forces are mainly dependent of the traveled distances.

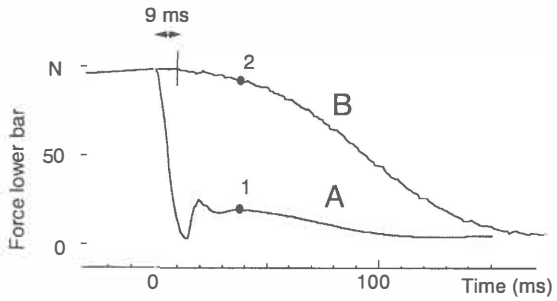


Figure 7. Average force recording (curve A, 100 N unloading experiment, travel distance 4.5 mm), and the decline in isometric force of the masseter muscles due to a silent period of 9 ms (curve B). Note that the maximum in residual force trace (curve A, point 1) is reached at an instant that the declining force of the jaw-closing muscles still has a level of 90% of their initial value (curve B, point 2).

of the force-length curve). This results in an increase (rather than a decrease) in isometric muscle force at jaw closure.

We calculated that the optimal sarcomere length would be reached at about 16 mm mouth opening (Fig. 4). This is in agreement with Mackenna & Türker (1983), who showed that in man maximal bite forces can be exerted at about 17 mm mouth opening. This implies that the force-length properties of the jaw-closing muscles can only contribute to a reduction in residual force at mouth openings smaller than 16 mm.

However, even with unloadings starting at a mouth opening of 16 mm or less, force-length properties cannot explain the considerable loss in bite force as found in our experiments since changes in sarcomere length are very small. For example, unloading at an initial mouth opening of 16 mm and a distance of travel of the mandible of 4.5 mm would change the sarcomere length about 0.15 μm, giving a decrease in isometric active force of about 10%.

Concerning the jaw-opening muscles a comparable way of reasoning can be given. At initial mouth openings used in our unloading experiments the sarcomeres of the jaw-opening muscles are below their optimum length (T.M.G.J. van Eijden, personal communication), so that they increase their force when stretched after the unloading. This gives an increase in force in the opening direction which contributes to a decrease in the bite force. However, as argued above, the effects of the force-length properties of the jaw-opening muscles may be counteracted by an upward movement of the hyoid bone.

Force-velocity properties and tendon creep

Previous studies (Slager et al., 1995; Nagashima et al., 1997) show that the decline in force in the dynamic phase after the unloading is largely due to the force-velocity properties of the jaw-closing muscles which lose a fair amount of their force when they shorten. Theoretically, this phenomenon could partly be responsible for the observed small value of the residual force: due to the in series arrangement of tendon sheets and muscle fibers in the jaw-closing muscles, and the long length of their tendinous sheets (about 50% of the total length of the muscles; Van Eijden et al., 1992, 1995 and 1996), the end of the jaw movement will not coincide with the end of the movement of the muscle fibers. Therefore, after the jaw is arrested, the muscle fibers will

continue to shorten (until they reach their final shortened state), contracting in series with the tendinous sheets (i.e. tendon creep) and loosing their force. However, this process will only give an additional reduction in force at the beginning of the post-dynamic phase, leaving the late low maximum in residual force unexplained.

Possible other explanations

In the cat (Joyce et al., 1969), in the frog (Edman et al., 1975, 1980 and 1993) and in the mouse (Ekelund & Edman; 1982), it was shown that when an active (stimulated) vertebrate muscle is stepwise shortened, a drop in force occurs which cannot be attributed to the force-length properties of the muscles alone; as Ekelund & Edman (1982) state: "...the muscle's ability to produce force seems to be decreased". The recovery of the (still stimulated) muscle is very slow: it takes 800-900 ms for a single frog muscle fiber to reach its original level of contraction (Edman, 1975 and 1980). The magnitude of the drop in force seems only sensitive to the degree of shortening of the muscle fiber: the larger the fiber shortening, the larger the drop in force (Edman, 1975). This is in line with our main experimental result that the relative magnitude of the residual force is largely dependent on the distance of travel of the mandible.

Edman (1975) and Edman et al., (1993) suggest that the extra drop in force is brought about by a non-uniform sarcomere length distribution over the length of a muscle fiber when contracting, or by a change in the myofilament system induced by a sudden shortening leading to a reduced actin-myosin interaction. This possibly also holds for our observations.

Conclusions

By exclusion we conclude that factors as reflex events, co-contraction, force-length and force-velocity properties of the jaw muscle cannot be responsible for the observed small values of the maxima of the residual forces as measured when the mandible is arrested shortly after the unloading of the jaw. In other words, explanations for the small values of the residual forces can neither be found at the level of the muscle-bone-connective-tissue-system of the oral system nor at the level of the individual muscles. Only poorly understood processes at the level of the sarcomeres remain to solve the riddle.

Acknowledgements

We are grateful to Mr. J. Mast and Mr. K. Vaartjes for designing and building the unloading apparatus and for their help during the experiments. We thank Mrs. C.A.M. Müller-Van den Berg for delivering the Phyaction 787 S/N 21252 stimulator.

5

Human bite force responses after fast and slow jaw-closing movements

Summary

In unloading experiments (in which experimentally the resistance to a forceful bite is suddenly removed) high bite forces decrease quickly as soon as the mouth starts closing. This quick decrease in force can be attributed to the force-velocity properties of the jaw closing muscles. When the mandible is arrested shortly after the unloading, the remnant bite forces (hereafter 'residual forces') -as measured when the jaw system is in static conditions again after the impact- also are small. The physiology underlying this phenomenon is not completely understood; it can not be explained by reflex events, nor by effects of co-contraction of the jaw-opening muscles nor by the active force-length properties of the jaw muscles. The force-velocity properties of the jaw closing muscles may partly be responsible for the low value of the residual force. The present paper concentrates on the influence of the velocity of jaw closure on the magnitude of bite force both during and after the jaw movement. We performed unloading experiments of a 100 N static bite at an initial mouth opening of 23.5 mm over four different distances of jaw closure. These experiments were performed twice, with and without the application of a damping resistance to jaw closure. The maximal velocity of jaw closure was reduced by a factor of five in the slow jaw closing movement in comparison with the fast ones. It is shown that after unloading the magnitude of the bite force during jaw movement is largely dependent on the velocity of jaw closure, whereas the magnitude of the residual force is insensitive to variations in closure velocity. It is submitted that the low residual forces are due to history-of-shortening-dependent changes in the contractile system.

Introduction

Unloading experiments of the jaw - in which experimentally the resistance to a forceful bite is suddenly removed - illustrate that high bite forces decrease quickly as soon as the mouth starts closing (Miles & Wilkinson, 1982; Van Willigen et al., 1997). For example: unloading a static bite of 100 N causes the bite force to fall to about 5 N within 14 ms after a jaw closure of 5 mm. Nagashima et al. (1997) and Slager et al. (1997) give evidence that the steep decline in bite force observed during the jaw movement is largely due to the force-velocity properties of the jaw-closing muscles: they lose a fair amount of their force when they shorten (shortening fibers produce less force than isometric ones), resulting in vanishing of the bite force.

When the mandible is arrested shortly after the unloading, the remnant bite forces (hereafter 'residual forces') -as measured when the jaw system is in static conditions again after the impact- also appear to be remarkably small (Van Willigen et al., 1997). For example: when arresting the mandible after a distance of travel of 5.0 mm after unloading a 100 N bite, the residual force is only 18 N.

In chapter 4 it is shown that the value of these residual forces is largely dependent on the distance of travel of the jaw before it is arrested (large travel distances give small residual forces) and is barely sensitive to variations in initial mouth opening. The percentage in loss of bite force appears to be independent of the magnitude of the initial bite. For example: a bite force of 100 N as well as a bite force of 40 N are both reduced to 60 % of their initial value after a distance of jaw travel of 1.5 mm, and to 25 % of their initial value after 4.5 mm jaw travel.

The physiology underlying the small values of the residual forces is still poorly understood. The small values can not be explained by reflex events (Hannam et al., 1968; Lamarre & Lund, 1975; Miles & Wilkinson, 1982; Yoshida & Inoue, 1995; Van Willigen et al., 1997) nor by effects of co-contraction of the jaw-opening muscles (Miles & Wilkinson, 1982; Miles & Madigan, 1983; Van Willigen et al., 1997) nor by the active force-length properties of the jaw muscles (MacKenna & Türker, 1983; Slager et al., 1998). For an extensive discussion see chapter 4.

In principle, the low value of the residual force may partly be explained by the force-velocity properties of the jaw-closing muscles. After the jaw is arrested, the muscle fibers of the jaw closing muscles may still keep on shortening for a while. This phenomenon is due to the in series arrangement of the muscle fibers and the tendinous sheets and to differences in their mechanical properties. As long as these internal movements occur (tendon creep) this will give a loss in force -due to the force-velocity properties of the muscle fibers- especially immediate after the jaw movement. The question remains whether the loss in force is both sufficient and long-lasting enough to explain the observed losses.

Also poorly understood processes at the level of sarcomeres may possibly be responsible for the low magnitude of the residual force (see chapter 4).

In the present paper unloading experi-

ments are discussed dealing with the effect of changes in velocity of jaw closure on the reduction in bite force in the dynamic phase of the experiments (as measured at the moment of impact when the mandible is stopped at various distances of jaw travel), and on the magnitude of the residual bite force in the static phase after the jaw movement. The experiments comprised unloading of a static bite of 100 N and arresting the mandible at a jaw closure of 1.0, 2.0, 3.0 or 4.0 mm thereafter. By varying the damping resistance, the velocity of jaw closure was varied.

If the force-velocity properties of the jaw closing muscles are responsible for the steep decline in bite force during jaw movement and for the small value of the residual force, we expect during slow jaw closing movements a slow decline in bite force in the dynamic phase. Furthermore, we expect an increase in residual force in the static phase after the jaw movement, due to a less pronounced tendon creep.

Material and methods

Participants

Seven dentate individuals participated in the experiments. All were free of signs and symptoms of muscular or temporomandibular dysfunction. The participants were fully informed about the experimental conditions. They gave their informed consent and participated in a protocol that complied with Dutch law.

Throughout the experiments the participants sat in front of an unloading device as illustrated in Figure 1. The position of the device was adjusted in height, so that it was at the level of the mouth. Bite forces were exerted between the upper and lower incisors and cuspids.

Unloading device

The device contained two parallel aluminum bars (80 mm long, 50 mm wide and 6 mm thick). For comfort the bite areas of the bars were covered with a thin layer of rubber. The upper bar was attached to two vertical plates mounted on a ground plate. The lower bar was fixed to a horizontal axis, which was connected to the vertical plates by means of two ball-bearings. Both lower bar and axis could rotate together relative to the rest of the device. The angular rotation of the lower bar was recorded with a magneto-resistive-sensor module (Philips KM11OBH/21), which was fixed to the axis. From the angle of rotation, the displacement of the bitten end of the lower bar could be calculated.

According to the method of Stegenga (1991), two pairs of balanced strain gauges were attached on either side of the lower bar 25 mm from the mouth piece, measuring the bite force applied to the lower bar independently of the point of force application.

The initial resistance to closing, before unloading, was achieved by an empowered solenoid of which the core was fixed to the lower bar by means of a steel cable; the solenoid itself was fixed to the body of the device. Thus the movement of the lower bar was prevented by the core, being held in place by the magnetic field of the solenoid. The magnetic field of the solenoid could be switched off either by hand or triggered by

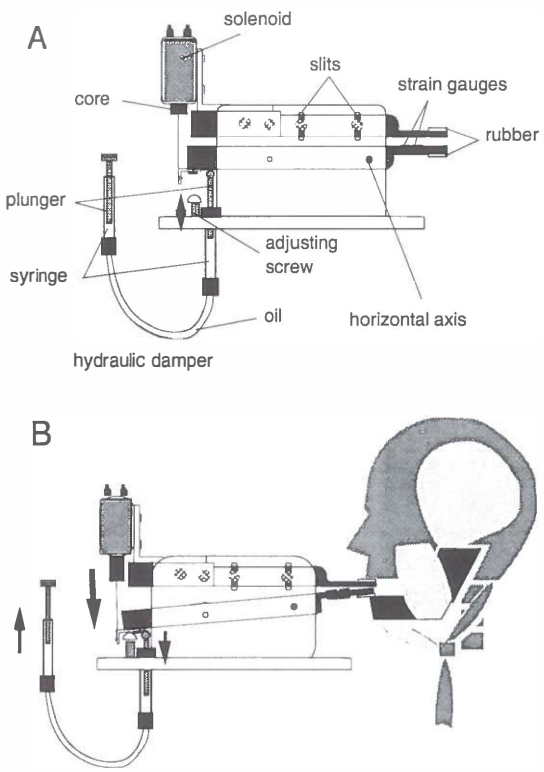


Figure 1. Unloading device: two parallel biting bars (the lower bar with strain gauges) are attached to two vertical plates mounted on a heavy support. The lower bar is fixed to an axis of which the angular displacement is measured. The initial resistance to closing is achieved by a solenoid. (A) Start of a jaw closing movement. To activate the hydraulic damping system, the plunger of the syringe mounted on the heavy support is brought in contact with the back of the lower bar (damped mode). (B) End of a jaw-closing movement.

the output of the strain gauges at a voltage equivalent to a (bite) force of 100 N.

When the solenoid was switched off the resistance of the unloading apparatus to a (bite) force applied on the bitten end of the lower bar suddenly disappeared, resulting in an acceleration of the lower bar; as a result the lower bar dropped at the back and its front was lifted (hereafter: UNDAMPED MODE).

The velocity of jaw closure could be altered by varying the damping resistance to the bite after unloading. This was achieved by bringing a hydraulic damper in contact with the back of the lower bar. This damper system consisted of two hypodermic syringes (ø 15 mm), connected to both ends of a flexible silicone tube (ø 6 mm); the system was filled with oil. One of the syringes was mounted through the ground plate at the level of the back of the lower bar. To bring the damper in action, the plunger of this syringe was brought in contact with the lower bar (Fig. 1A) by pushing down the plunger of the other syringe (hereafter: DAMPED MODE). The damp-

ing characteristics of the hydraulic damper were such, that with an almost constant upward force of 100 N (applied by means of good quality long elastic bands) the maximal velocity of the lower bar after unloading was reduced by a factor of 4 in comparison with the undamped mode after a distance of travel of 4 mm (see paragraph controls and Fig. 2).

The position of the upper bar was adjusted by means of slitted holes so that the initial mouth opening of the subjects at the start of each experiment was 23.5 mm when biting on the device. The mouth opening of the subjects was defined as the interincisor distance added to the vertical overlap of the dentition (overbite).

The distance of travel of the bitten end of the lower bar could be varied by means of an adjusting screw which was mounted through the ground plate near the end of the lower bar (Fig. 1). To buffer the shock of collision, the head of the screw was covered with a soft plastic cap.

Protocol

With the apparatus in the undamped or damped mode, the subjects were asked to bite through a resistance of 100 N with care. This was done at an initial mouth opening of 23.5 mm and a distance of travel of the mandible of 1.0, 2.0, 3.0 or 4.0 mm after unloading. This gave 8 experimental conditions. All experiments were repeated 5 times giving a total of 40 observations per subject.

Controls

The mechanical properties of the unloading apparatus in the undamped mode were established in a series of tests described in previous studies (Van Willigen et al. 1997; Nagashima et al. 1997).

The mechanical properties of the un-

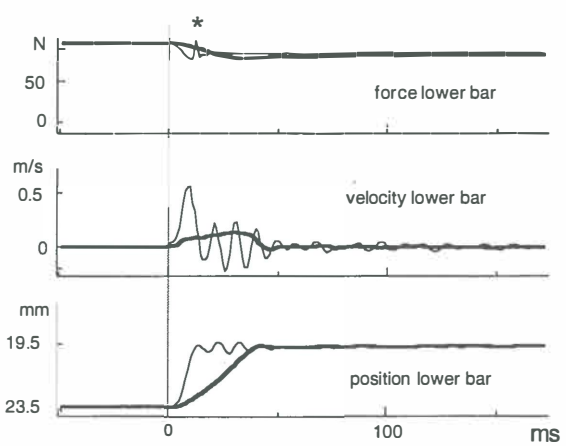


Figure 2. Superimposed force, (calculated) velocity and position recordings of the lower bar when the apparatus was loaded with an (almost) constant force of 100 N and the solenoid was switched off by hand (thick lines, damped mode; thin lines, undamped mode). The lower bar started to move at time 0. The velocity was reduced 4.3 times in the damped mode as compared to the undamped mode. Asterisks, rebound reaction force after the back of the lower bar came into collision with the adjusting screw.

loading apparatus in the damped mode were measured by loading the lower bar with 100 N in an upward direction [by means of a number of long elastic bands (total stiffness 3.0 N/mm)], and switching off the solenoid by hand. The outcome was compared with similar observations in the undamped mode.

Data analysis

The results of each of the experimental conditions were pooled across subjects and averaged. Figure 3 depicts averages of the force (upper trace) and position recordings (bottom trace) of two 100 N unloading experiments (undamped mode Fig. 3A; damped mode Fig. 3B), with a distance of travel of 3.0 mm (n=35, standard deviation in grey), and of the calculated average velocity (middle trace). The velocity of the lower bar/lower teeth combination was calculated from the position recordings of the bitten end of the lower bar by differentiation of the position signals over time.

The moment of unloading (at time 0) was defined as the moment that the bite force dropped (Fig. 3, top panels, point 1).

A non zero force recording implies that during the experiment the teeth are in contact with the lower bar and the time of impact of the lower bar is the same as that of the teeth. This time of impact can be clearly seen in the force recordings of the undamped experiments. However, in the damped experiments often no clear change in the force signal related to the impact can be established.

Therefore, we looked in the *undamped* experiments for the time of occurrence of the first minimum in the force signal after unloading (open arrow in the force display of Fig. 3A), and took the value in the force and position signal at this instant. [This point coincides with the first moment of contact of the back of the lower bar with the plastic cap covering the adjusting screw. After this instant the bite force increases.] The value found in the force signal was taken as 'impact force' in the undamped experiments. The value of the position signal (point 2 in the position displays of Fig. 3) was used to find the value of the impact force (i.e. after the same distance of travel) in the analogous *damped* experiments (dark arrow in the force display of Fig. 3B).

We defined the 'dynamic phase' of the experiment as the time span between unloading and impact (Fig. 3 in grey), and the 'post dynamic phase' as the period after impact of the lower bar (Fig. 3). Forces measured in the latter period were called 'residual forces'.

In order to quantify the residual forces, we determined inside a time window of 15-60 ms after the moment of impact the maximum in the residual force (point 4 in the top panel of Fig. 3A). The boundary of 15 ms was taken to exclude transient force peaks caused by the collision of the back of the lower bar with the head of the adjusting screw after impact (point 3 in the top panel of Fig. 3A).

In the damped mode at distances of travel larger than 2.5 mm, the force declined continuously so that there was no maximum in the residual force. Only a small bump in the declining force could be identified. In the absence of a maximum in the damped mode, we took the maximum in the first derivative as the residual force (point 5 in the top panel of Fig. 3B).

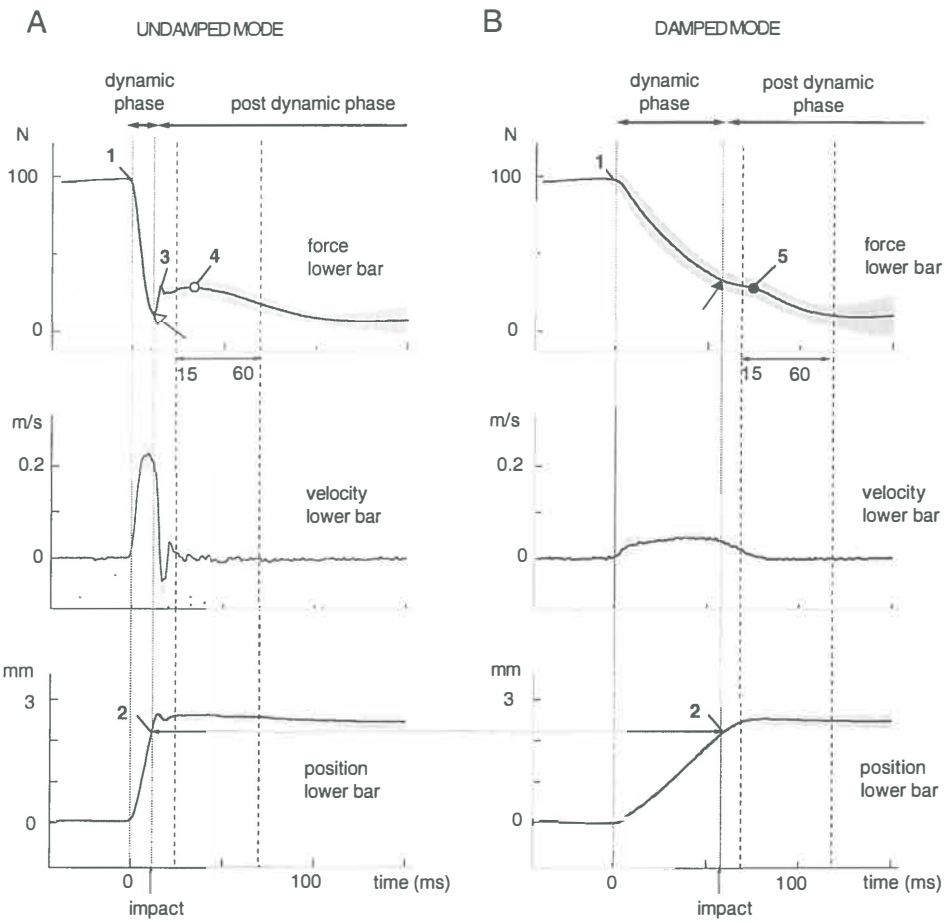


Figure 3. Illustration of the outcome of an analysis, as described in the Material and Methods. Averages of force (upper trace) and position recordings (the bottom trace) of two 100 N unloading experiments with a distance of travel of 3.0 mm (undamped mode (A); damped mode (B), n=35, SD in grey), and the calculated average velocity (middle trace). (1) Moment of unloading and magnitude of the bite force at unloading; (2) position of the lower bar at the time of impact; open and dark arrow, time of impact and magnitude of impact force; (3) transient peak force due to collision of lower bar to head of adjusting screw; (4) maximum in the residual force; (5) maximum in the first derivative of the residual force; open and dark circle, time of occurrence and magnitude of the residual force as defined in this study.

The residual forces are indicated by an open circle in the undamped mode (Fig. 3A, 4 and 5) and a dark circle in the damped mode (Fig. 3B, 4 and 5).

The time elapsed from unloading to impact was called t-Fimpact and the time from unloading to residual force t-Fresidu.

All processing was done on an Apple Macintosh IIx computer system extended with a Rocket 33 accelerator board and running 'DataMonster 2.0', an analytical program written by one of us (E.O.).

Results

Mechanical properties of the unloading device in the damped mode

Figure 2 gives the output of the lower bar force transducer, the (calculated) velocity of the lower bar and the position signal when the unloading apparatus was loaded with an upward force of 100 N generated by a group of long elastic bands, and the solenoid switched off by hand (thick lines: damped mode, thin lines: undamped mode). The distance of travel of the lower bar was 4.0 mm, the initial distance between the bars was 23.5 mm. The lower bar started to move at time 0.

As can be seen the velocity of the lower bar was reduced considerably (4.3 times) in the damped mode as compared to the undamped mode. [Undamped mode: time of travel of the lower bar 13 ms and impact velocity 0.56 m/s. Damped mode: time of travel of the lower bar 34 ms and impact velocity 0.13 m/s.]

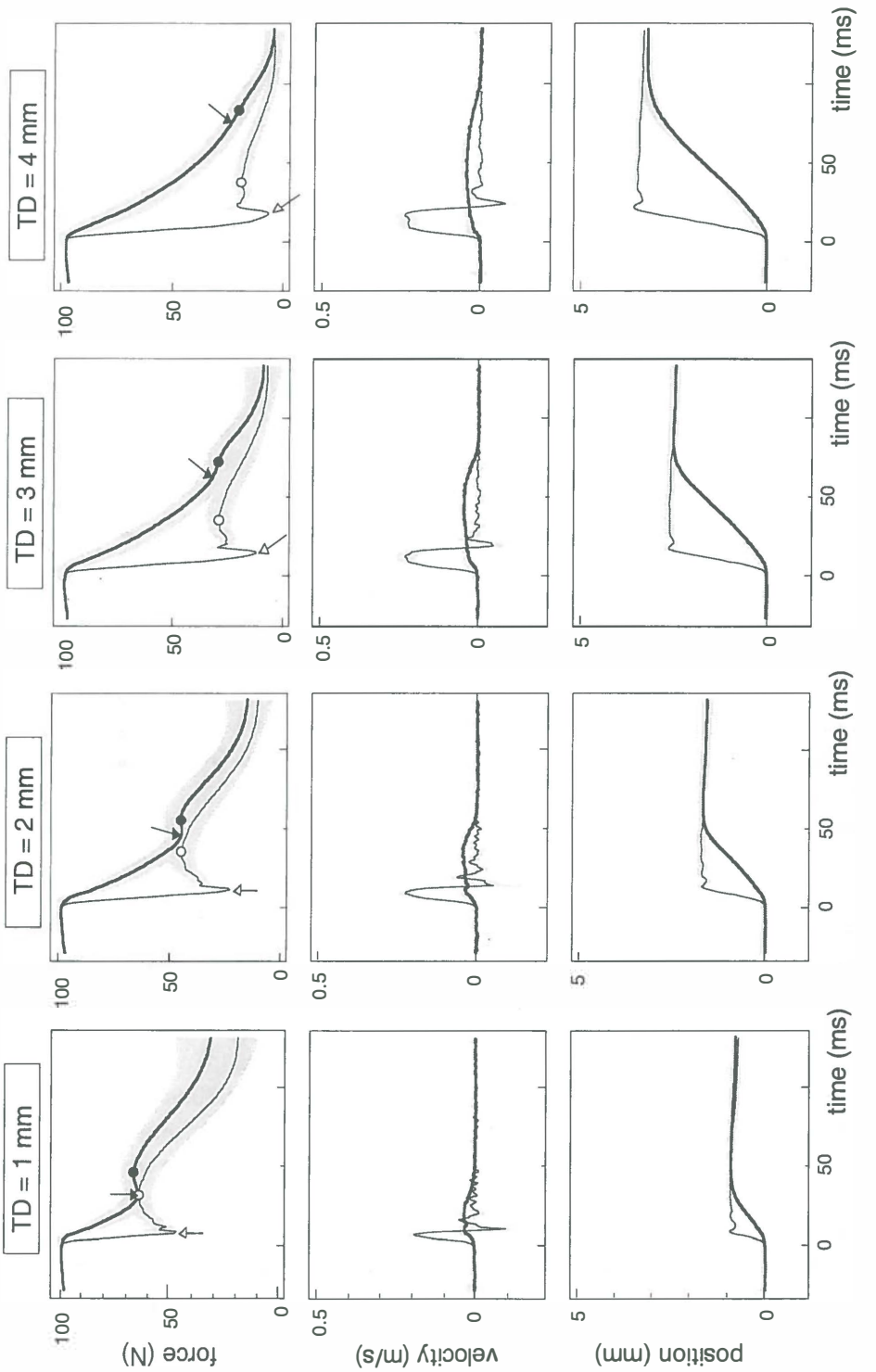
In the undamped mode there was a rebound oscillation after the back of the lower bar came into collision with the adjusting screw; this oscillation subsided in 3 to 4 cycles (Fig. 2, velocity panel, thin line). The rebound reaction force can be seen as a small hump in the force trace (Fig. 2, asterisk in force panel).

The experiments

Figure 4 displays the averages of the pooled data across subjects and their standard deviations (n=35) for the 8 experimental conditions. It depicts the averaged force, velocity and position recordings of the experiments in the undamped mode (thin line; open arrow: impact force; open circle: residual force) and in the damped mode (thick line; dark arrow: impact force; dark circle: residual force). The initial bite force was 100 N; distances of travel were 1.0, 2.0, 3.0 and 4.0 mm, respectively.

The outcome of the experiments were remarkably reproducible, as can be seen from the small standard deviations (grey bands). In all the experimental conditions the dynamic phase lasted considerably longer in the damped mode as compared to the undamped mode (Fig. 4; t-Fimpact in Table1). As a consequence, the maximal

Figure 4. Results of 280 experiments (seven subjects, five trials per condition, n=35, 8 experimental conditions). The panels depict forces, velocities and positions of the lower bar of the unloading experiments in the damped mode (thick line) and in the undamped mode (thin line). The time of occurrence and the magnitude of the residual forces are indicated by circles (open circles, undamped mode; dark circles, damped mode), the impact forces by arrows (open arrows, undamped mode; dark arrows, damped mode).


TD = 1 mm
TD = 2 mm
TD = 3 mm
TD = 4 mm
force (N)
velocity (m/s)
position (mm)
time (ms)
100
50
0
0.5
5
0
50

MODE	TD (mm)	Fimpact (N)	t-Fimpact (ms)	Fresidu (N)	t-Fresidu (ms)	vmax (m/s)
undamped	1.0	47 ± 8	8	63 ± 6	34	0.19 ± 0.02
damped	1.0	64 ± 6	31	66 ± 5	45	0.04 ± 0.01
undamped	2.0	22 ± 4	12	45 ± 6	33	0.22 ± 0.02
damped	2.0	45 ± 7	45	45 ± 7	56	0.04 ± 0.01
undamped	3.0	11 ± 3	15	29 ± 6	37	0.23 ± 0.03
damped	3.0	31 ± 6	67	29 ± 6	74	0.05 ± 0.01
undamped	4.0	6 ± 2	17	19 ± 4	37	0.24 ± 0.04
damped	4.0	20 ± 5	84	20 ± 5	83	0.05 ± 0.01

Table 1. Moment and magnitude of the impact force (Fimpact) and the residual force (Fresidu) and the maximal velocity (vmax) of jaw closure in all experimental conditions. TD = travel distance of the lower bar; t-Fimpact, time span from unloading to impact force; t-Fresidu, time span from unloading to residual force.

velocity (vmax) of the mandible/lower bar combination was about 5 times smaller in the damped mode (Fig. 4: velocity displays; vmax in Table 1).

When comparing the force profiles in the dynamic phase, it can be seen that the bite force declined more steeply in the undamped mode as compared to the damped mode (Fig. 4: force displays). For example, after a distance of travel of 2.0 mm, in the undamped mode the 100 N bite force has 45 N (sd 7 N) left; the jaw had than travelled 45 ms (Fimpact and t-Fimpact in Table1).

The relationship between the impact forces in both modes and the amount of jaw closure is also clearly illustrated in Figure 5A. It can be seen that the magnitude of the impact force in the undamped mode is considerably larger than in the damped mode.

In contrast to the large differences found between force, position and velocity profiles in the dynamic phase of the two types of experiments, a large similarity exists in the magnitude of the residual forces (Figs. 4 and 5B, and Table 1). Figure 5B clearly illustrates that the values of the residual forces appear to be insensitive to the velocity of jaw closure; the magnitudes of the residual forces have overlapping bands of standard deviation.

Only a shift in the time of occurrence of the residual force (force displays in Fig. 4) could be detected between the experiments. For example: in the undamped mode, after a travel distance of 2.0 mm, the residual force is 45 N (sd 6 N, n=35) and t-Fresidu 33 ms. In the damped mode we find the same figure for the residual force (45 N, sd 7 N, n=35) but now at a t-Fresidu of 56 ms

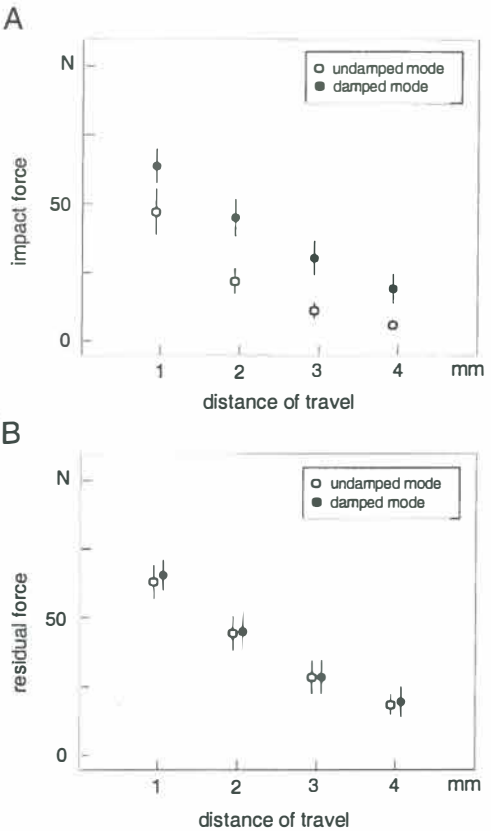


Figure 5. (A) Plot of the average impact force as function of the distance of travel of the lower bar. (B) Plot of the average residual force as function of the distance of travel of the lower bar. Open circles, undamped mode; dark circles, damped mode. Note that -in contrast with the magnitude of the impact forces- the residual forces are insensitive to the velocity of jaw closure.

(Table 1).

Discussion

Decline in force in the dynamic phase after unloading

In the present experiments, we have shown that after an unloading of a static bite the decline in bite force in the dynamic phase strongly depends on the velocity of jaw closure. Fast jaw-closing movements are accompanied by a rapid decay of bite force, whereas slow jaw closures are associated with a slow decline in bite force.

The results are in good agreement with our hypothesis (Nagashima et al., 1997; Slager et al., 1997) that especially the force-velocity properties of the jaw closing muscles are responsible for the decline in bite force during jaw closing movements. This hypothesis is based on a numerical forward dynamic model of the human jaw system, simulating unloading experiments (as described in chapter 3). With this model we illustrated that, due to their force-velocity properties, and without any neural control, the muscle fibers of the jaw closing muscles can lose all their force over a distance of jaw travel of only 4.5 mm when unloading a 100 N static bite and the jaw is allowed to close freely.

The influence of the force-velocity properties on the decline in bite force is also reflected in the unloading experiments with a damper. Here -as compared to the undamped conditions- the velocity of the muscle fibers of the jaw closing muscles is strongly reduced during the dynamic phase of the experiment and so is the reduction in bite force during jaw movement.

The experimental results also indicate that the reduction in bite force in the dynamic phase is not caused by the force-length properties of the jaw muscles or by the level of co-contraction of the jaw-opening muscles, since these influences were

equal in both modes.

Residual force in the post dynamic phase after unloading

In contrast to the loss in bite force in the dynamic phase after unloading, the large loss in bite force in the post-dynamic phase is still poorly understood.

As discussed in chapter 4, reflex events can not be responsible for the small values of the residual forces. Due to the electromechanical delay, the silent period in the jaw-closers and the reflexes in the jaw-openers (both being part of the motor control of the lower jaw and evoked by the sudden jaw movement) are incapable of inducing the small and early residual forces. They can induce only a loss of 10 % of the initial bite force (Slager et al., 1998).

Also the contribution of the force-length properties of the jaw-opening muscles to the reduction in bite force is probably small, because the level of co-contraction is low (Miles & Wilkinson, 1982; Miles & Madigan, 1983; Van Willigen et al., 1997; Yoshida & Inoue, 1995) and the human jaw-opening muscles are weak (Sharkey et al. 1984). Furthermore, possible effects of the force-length properties of the jaw-opening muscles may be counteracted by movements of the hyoid bone if it moves in synchrony with the mandible after unloading as during jaw closure (Thexton et al., 1981; Pancherz et al., 1986).

The force-length properties of the jaw closing muscles contributed even negatively to the reduction in bite force. At the degrees of initial mouth opening used in our experiments the sarcomeres of the jaw closing muscles are beyond their optimum length (Van Eijden & Raadsheer, 1992; Van Eijden et al., 1995 and 1996). This even gives an increase in isometric active force at jaw closure (for further discussion see chapter 4).

In this paper, we studied the influence of the velocity of jaw closure on the magnitude of the residual bite force in unloading experiments. The experimental results show no difference in residual force in the damped and undamped conditions. This suggests that effects of the force-velocity properties in conjunction with tendon creep are not long lasting enough to account for the small residual bite forces.

Unknown muscle physiological properties responsible for low residual force

Apparently, a muscle property not taken into account is responsible for the low magnitudes of the residual forces.

It is shown that the low magnitude of the residual force is unrelated to the velocity at which the jaw traveled to its new position. It is however strongly related to the history of jaw travel (Fig. 5B). Furthermore, the loss in force in the post-dynamic phase is activation dependent (chapter 4): the percentage in loss of bite force is independent of the magnitude of the initial bite force. So the muscle property we are looking for is sensitive to the level of activation and the amount of muscle shortening, but not to the history of velocity of shortening.

We speculate that this unidentified muscle property is located in the active contractile system of the jaw-closing muscles. In concurrence with Edman (1975) and Edman et al. (1993) we suggest that the loss in force is due to a reduced actin-myosin interaction after shortening, or a non-uniform sar-

comere distribution build up over the length of muscle fibers.

Functional interpretations

Our observations of the relationship between the loss in force and the velocity of jaw closure after unloading can be interpreted as being useful for biting through various types of food.

When biting through hard and brittle food, like peanut or carrot, high bite forces are required which have to diminish abruptly after breaking of the food; otherwise the velocity at which the teeth will collide might cause damage of the dental elements. This is in line with our observations in the undamped experiments: after unloading a strong bite, a fast jaw closure is associated with a rapid decrease in force during jaw movement, resulting in a low impact velocity of the teeth. This happens without a change in motor drive to the closing muscles.

The slow decay of bite force associated with slow jaw-closing movements is functional for biting through tough food, like gum or meat: if the bite force would vanish quickly with slow closing, we would not be able to bite through the food at any useful velocity. Due to the resistance of this kind of food there is no danger of dental damage, although bite forces are still high: the acceleration of the mandible is kept low and therefore its velocity, as shown in the experiments in the damped mode.

So a build up of jaw velocity is either prevented by muscle properties in the case of brittle food or by the food itself in the case of tough food.

Acknowledgements

We are grateful to J. Mast and K. Vaartjes for designing and building the unloading apparatus and for their help during the experiments.

Reflections

Movements are a part of daily life. They are often complex and hard to understand in terms of biomechanics and neural control. Fundamental knowledge on how these movements are performed may be helpful, for instance in rehabilitation or in sport.

In this thesis we have chosen to investigate one specific movement, which occurs when the resistance to a static force is suddenly withdrawn. In daily life we make these kinds of movements when pulling a cork from a bottle of wine, when breaking a branch, when pushing against a door which suddenly yields, or when biting through hard food, which suddenly breaks.

We investigated the latter movements in a controlled way; we carried out unloading experiments of the jaw, in which experimentally the resistance to a forceful static bite was suddenly withdrawn. We found that the bite force rapidly declined after the unloading, resulting in a small velocity of the lower front teeth at impact. Moreover, when the lower jaw is stopped in its movement shortly after unloading, the remnant bite force appeared to be remarkably small.

We have shown that

1) *The rapid decline in bite force in the dynamic phase after unloading of a bite is due to the force-velocity properties of the jaw-closing muscles and is not caused by neural control. The activation dependent mechanical properties of the jaw muscles are helpful in avoiding damage.*

We suggest that this also holds for other motor tasks; the force-velocity properties of active muscles catch up unexpected perturbations during movement at a very early stage where neural processing is too slow to handle the instances of perturbations.

2) *The muscle physiology underlying the small residual forces in the post-dynamic phase after an unloading is poorly understood. The small values of the residual forces are not due to the force-length properties of the jaw muscles or to neural control. They are due to a muscle property which is coupled to the level of activation and the amount of muscle shortening, but not to the history of velocity of shortening. We speculate that the loss in muscle force finds its origin in one of the processes involved in force generation at the level of sarcomeres.*

We conclude that the stiffness of the jaw closing muscles is extremely high (there is a large loss in force over small distances of jaw travel) and activation dependent (the loss in relative bite force in the post-dynamic phase is independent of the magnitude of the initial bite force).

This high stiffness is not only useful in handling unexpected perturbations during movement, but is also helpful in maintaining posture. For instance, during walking and running the position of the jaw has to be maintained, otherwise the lower jaw jumps up- and downward excessively during each step. With a high muscle stiffness, the posture of the jaw is easier to maintain.

The high stiffness of the jaw muscles is not a property which is unique for these muscles only: it is also found in other skeletal muscles. For example, we also found large losses in force in the intrinsic hand muscles after a static unloading task of the

fingers (i.e. a 'scissors-like movement', in which the middle finger moved to the fore-finger (unpublished observation).

Apparently, more tasks benefit from the same principle.

3) *The large losses in force in the dynamic and post-dynamic phase after unloading are not due to co-contraction of the jaw-closing and -opening muscles. The level of co-contraction was always low during the unloading experiments.*

It is easy to stiffen the jaw by increasing the level of co-contraction, but it seems hard to exert high bite forces with a high level of co-contraction simultaneously. This may be due to a neural constraint. For the control of impact velocity this is not a favorable constraint (a computer model showed that co-contraction helps to limit the impact velocity).

However, it is shown in this thesis that a high level of co-contraction is not necessary to handle unexpected perturbations of the jaw. Only activation of muscles seems a precondition for handling perturbations. Due to the activation of muscles (here: the jaw-closing muscles) the activation dependent mechanical properties of the jaw-closing muscles give a strong reduction in bite force.

During maintenance of posture, hardly any muscle activity is neccessary. Only subtle alternations of agonist and antagonist activity are needed. When during maintenance of posture external perturbations are expected, a high level of co-contraction seems neccessary to secure the integrity of the human body. This may be particularly relevant in fast action sports.

4) *After unloading of the jaw, changes in length of the muscles are not equal to changes in length of the muscle fibers, due to compliance of the tendinous sheets.*

This phenomenon is almost impossible to measure in humans. It is of interest, however, to note that with our firmly tested computer model, it can be easily shown. This is a nice example of the useful interaction between an experimental design and a computer simulation.

References

Angel RW, Eppler W and Iannone A (1965). Silent period produced by unloading of muscle during voluntary contraction. *J. Physiol.* 180, 864-870.

Bellemare F, Woods JJ, Johansson R and Bigland-Ritchie B (1983). Motor-unit discharge rates in maximal voluntary contractions of three human muscles. *J. Neurophysiol.* 50, 1380-1392.

Buchanan TS and Lloyd DG (1995). Muscle activity is different for humans performing static tasks which require force control and position control. *Neurosci. Letts* 194, 61-64.

Close R (1964). Dynamic properties of fast and slow skeletal muscles of the rat during development. *J. Physiol. (London)* 173, 74-95.

De Jonge H (1963). *Inleiding tot de medische statistiek deel 1.* Wolters-Noordhoff, Groningen.

Dijkstra PU, de Bont LGM, Stegenga B and Boering G (1995). Temporomandibular joint mobility assessment: a comparison between four methods. *J. Oral. Rehab.* 22, 439-444.

Dufossé M, Hugon M and Massion J (1985). Postural forearm changes induced by predictable in time or voluntary triggered unloading in man. *Exp. Brain Res.* 60, 330-334.

Edman KAP (1975). Mechanical deactivation induced by active shortening in isolated muscle fibres of the frog. *J. Physiol.* 246, 255-275.

Edman KAP (1980). Depression of mechanical performance by active shortening during twitch and tetanus of vertebrate muscle fibres. *Acta Physiol. Scand.* 109, 15-26.

Edman KAP, Caputo C and Lou F (1993). Depression of tetanic force induced by loaded shortening of frog muscle fibres. *J. Physiol.* 466, 535-552.

Ekelund MC and Edman KAP (1982). Shortening induced deactivation of skinned fibres of frog and mouse striated muscle. *Acta Physiol. Scand.* 116, 189-199.

Eriksson P-O, Eriksson A, Ringqvist M and Thornell L-E (1981). Special histochemical muscle-fibre characteristics of the human lateral pterygoid muscle. *Archs oral Biol.* 26, 495-507.

Eriksson P-O, Eriksson A, Ringqvist M and Thornell L-E (1982). Histochemical fibre composition of the human digastric muscle. *Archs oral Biol.* 27, 207-215.

Eriksson P-O, and Thornell L-E (1983). Histochemical and morphological muscle-fibre characteristics of the human masseter, the medial pterygoid and the temporal muscle. *Archs oral Biol.* 28, 781-795.

Falkenström CH (1993). *Biomechanical design of a total temporomandibular joint replacement.* (dissertation), Offset-drukkerij FEBO, Enschede.

Flitney FW and Hirst DG (1978). Cross-bridge detachment and sarcomere 'give' during stretch of active frog's muscle. *J. Physiol.* 276, 449-465.

Fuglevand AJ, Winter DA, Patla AE and Stashuk D (1992). Detection of motor unit action potentials with surface electrodes: influence of electrode size and spacing. *Biol. Cybern.* 67, 143-153.

Goodwin GM and Luschei ES (1974). Effects of destroying spindle afferents from jaw muscles on mastication in monkeys.

J. Neurophysiol. 37, 967-981.

Gordon AM, Huxley AF and Julian F (1966). The variation in isometric tension with sarcomere length in vertebrate muscle fibers. *J. Physiol.* 184, 170-192.

Grillner S (1972). The role of muscle stiffness in meeting the changing postural and locomotor requirements for force development by the ankle extensors. *Acta Physiol. Scand.* 86, 92-108.

Hannam AG, Matthews B and Yemm R (1968). The unloading reflex in masticatory muscles of man. *Archs Oral Biol.* 13, 361-364.

Hannam, AG and Langenbach GEJ (1995). Modelling the masticatory system during function. In: *Proceedings of the Osaka International Oral Physiology Symposium on Brain and Oral Function,* edited by T. Morimoto, T. Matsuya and K. Takada. Osaka: Osaka University, 217-226.

Hill AV (1938). The heat of shortening and the dynamic constants of muscle. *Proc. R. Soc. London Ser. B,* 126, 136-195.

Joyce GC, Rack PMH and Westbury DR (1969). The mechanical properties of cat soleus muscle during controlled lengthening and shorthening movements. *J. Physiol.* 204, 461-474.

Julian FJ and Moss RL (1981). Effects of calcium and ionic strength on shorthening velocity and tension development in frog skinned muscle fibres. *J. Physiol. (London)* 311, 179-199.

Kalunzy P and Wiesendanger M (1992). Feedforward postural stabilization in a distal bimanual unloading task. *Exp. Brain Res.* 92, 173-182.

Koolstra JH, van Eijden TMGJ, Weijs WA, and Naeije M (1988). A three-dimensional mathematical model of the human masticatory system predicting maximum possible bite forces. *J. Biomech.* 21, 563-576.

Koolstra, JH and Van Eijden TMGJ (1995). Biomechanical analysis of jaw-closing movements. *J. Dent. Res.* 74, 1564-1570.

Laboissière R, Ostry DJ and Feldman AG (1996). The control of multi-muscle systems: human jaw and hyoid movements. *Biol. Cybern.* 74, 373-384.

Lamarre Y and Lund JP (1975). Load compensation in human masseter muscles. *J. Physiol.* 253, 21-35.

Lieber RL, Leonard ME, Brown CG and Trestik CL (1991). Frog semitendinosis tendon load-strain and stress-strain properties during passive loading. *Am. J. Physiol.* 261, C86-92.

MacKenna BR and Türker KS (1978). Twitch tension in the jaw muscles of the cat at various degrees of mouth opening. *Archs oral Biol.* 23, 917-920.

MacKenna BR and Türker KS (1983). Jaw separation and maximum incising force. *J. Prosthet. Dent.* 49, 726-730.

McMillan AS and Hannam AG (1992). Task dependence of human masseter motor unit reflex behaviour. *Exp. Brain Res.* 88, 443-446.

Merlini L and Palla S (1988). The relationship between condylar rotation and anterior translation in healthy and clicking temporomandibular joints. *Schweiz Monatsschr. Zahnmed.* 98, 1191-1199.

Miles TS and Wilkinson TM (1982). Limitation of jaw movement by antagonist muscle stiffness during unloading of human jaw closing muscles. *Exp. Brain Res.*

46, 305-310.

Miles TS and Madigan ML (1983). Programming of antagonist muscle stiffness during masticatory muscle unloading in man. *Archs oral Biol.* 10, 947-951.

Miles TS, Nordstrom MA and Türker KS (1986). Length-related changes in activation threshold and wave form of motor units in human masseter muscle. *J. Physiol.* 370, 457-465.

Nagashima T, Slager GEC, Otten E, Broekhuijsen ML and Van Willigen JD (1997). Impact velocities of the teeth after a sudden unloading at various initial bite forces, degrees of mouth opening and distances of travel. *J.Dent. Res.* 76, 1751-1759.

Nelder JA and Mead R (1965). A simplex method for function minimization. Comput. J. 7, 308-313.

Nygaard E, Houston M, Suzuki Y, Jorgensen K and Saltin B (1983). Morphology of the brachial biceps muscle and elbow flexion in man. *Acta Physiol. Scand.* 117, 287-292.

Obwegeser HL, Farmand M, Al-Majali F and Engelke W (1987). Findings of mandibular movement and the position of the mandibular condyles during maximal mouth opening. *Oral surg, Oral med, Oral path.* 63, 517-525.

Otten E (1987a). A myocybernetic model of the jaw system of the rat. *J.Neurosci Methods* 21, 287-302.

Otten E (1987b). Optimal design of vertebrate and insect sarcomeres. *J. Mor*ph. 191, 49-62.

Otten E (1991). The control of movements and forces during chewing. In: *Feeding and the texture of foo*d, edited by J.F.V. Vincent and P.J. Lillford. Cambridge: Cambridge University Press, 123-141.

Pancherz H, Winnberg A, Westesson P (1986). Masticatory muscle activity and hyoid bone behavior during cyclic jaw movements in man. *Am J Orthod* . 89, 122-131.

Press WH, Flannery BP, Teukolsky SA and Vetterling WT (1986). In: *Numerical recipes*. Cambridge: Cambridge University Press, 277-282.

Pruim GJ, de Jongh HJ and ten Bosch JJ (1980). Forces acting on the mandible during bilateral static bite at different bite force levels. *J. Biomech.* 13, 755-763.

Rack PMH and Westbury DR (1974). The short range stiffness of active mammalian muscle and its effect on mechanical properties. *J. Physiol*, 240, 331-350.

Sharkey P, Boyle DK, Orchardson R and McGowan DA (1984). Jaw opening forces in human subjects. *Br. Dent. J.* 156, 89-92.

Slager GEC, Otten E, Van Willigen JD and Broekhuijsen ML (1995). Contribution of mechanical properties of jaw muscles to the control of unexpected jaw-closing movements. In: *Proceedings of the Osaka International Oral Physiology Symposium on Brain and Oral Function*, edited by T. Morimoto, T. Matsuya and K. Takada. Osaka: Osaka University, 369-373.

Slager GEC, Otten E, Van Eijden, TMGJ, and Van Willigen JD (1997). A mathematical model of the human jaw system simulating static biting and movements after unloading. *J. Neurophysiol.* 78, 3222-3233.

Slager GEC, Otten E, Nagashima T and Van Willigen JD (1998). The riddle of the

large loss in bite force after fast jaw closing movements. *J. Dent. Res.* (accepted)

Soechting JF and Lacquaniti F (1988). Quantitative evaluation of the electromyographic responses to multidirectional load perturbations of the human arm. *J. Neurophysiol.* 59, 1296-1313.

Stegenga B (1991). *Temporomandibular joint osteoarthrosis and internal derangement; diagnostic and therapeutic outcome assessment,* (dissertation). University of Groningen, Groningen.

Thexton A.J. and Hiiemae K.M. (1975) The twitch-contraction characteristics of opossum jaw musculature. *Archs oral Biol.* 20, 743-748.

Thexton AJ, McGarrick J and Wallace J (1981). Phase relationship between human jaw and hyoid movement. *J. Dent. Res.* 60, 475.

Trestik CL and Lieber RL (1993). Relationship between the achilles tendon mechanical properties and gastrocnemius muscle function. *J. Biomech. Eng.* 115, 3, 225-230.

Van Eijden TMGJ, Brugman P, Weijs WA and Oosting J (1990). Coactivation of jaw muscles: recruitment order and level as a function of bite force direction and magnitude. *J. Biomech.* 23, 475-485.

Van Eijden TMGJ (1991). Three-dimensional analyses of human bite-force magnitude and moment. *Archs oral Biol.* 36, 535-539.

Van Eijden TMGJ and Raadsheer MC (1992). Heterogeneity of fiber and sarcomere length in the human masseter muscle. *Anat. Rec.* 232, 78-84.

Van Eijden TMGJ, Koolstra JH and Brugman P (1995). Architecture of the human pterygoid muscles. *J. Dent Res.* 74, 1489-1495.

Van Eijden TMGJ, Koolstra JH and Brugman P (1996). Three-dimensional structure of the human temporalis muscle. *Anat. Rec.* 246, 565-572.

Van Willigen JD, Broekhuijsen ML, Melchior HJ, Karkazis HC, Kossioni A and Heath MR (1993). Digastric muscle responses as a function of knowledge of the task to be performed. *Archs oral Biol.* 38, 113-121.

Van Willigen JD, Slager GEC, Broekhuijsen ML and Otten E (1995). Digastric muscle responses as a function of knowledge of the task to be performed. In*: Proceedings of the Osaka International Oral Physiology Symposium on Brain and Oral Function,* edited by T. Morimoto, T. Matsuya and K. Takada. Osaka: Osaka University, 205-216.

Van Willigen JD, Otten E, Slager GEC and Broekhuijsen ML (1997). Contribution of the digastric muscles to the control of bite force in man. *Archs oral Biol.* 42, 45-56.

Walker SM and Schrodt GR (1974). Segment lengths and thin filament periods in skeletal muscle fibers of the rhesus monkey and the human. *Anat. Rec.* 178, 63-82.

Weijs WA and Hillen B (1985). Cross-sectional Areas and Estimated Intrinsic Strength of the Human Jaw Muscles. *Acta Morphol. Neerl.-Scand.* 23, 267-274.

Yoshida K and Inoue H (1995). Unloading reflex of masticatory muscles during comminution of food. In: *Proceedings of the Osaka International Oral Physiology Symposium on Brain and Oral Function*, edited by T. Morimoto, T. Matsuya and K. Takada. Osaka: Osaka University, 357-363.

Summary

This thesis deals with the events which underlay the rapid decline in bite force when the resistance to a static bite is suddenly withdrawn.

This is an important subject since bite forces can be very high (up to 600 N) so that there is a serious risk of damage to the dental elements when biting through hard and brittle food at these high forces because the effective mass of the lower jaw is small (only about 0.20 kg at the level of the front teeth) and the margin of safety limited (jaw separations during biting are in the order of 2 to 3 cm). For example, when biting through brittle food at an initial mouth opening of 2 cm with a constant bite force of 100 N, the teeth, theoretically, will collide with a velocity of 4.5 m/s (i.e. 16 km/h) after breaking of the food.

However, such high velocities do not occur in reality since in unloading experiments -in which, experimentally, the resistance to a forceful static bite is suddenly withdrawn- it is shown that bite forces decrease at a high rate as soon as the mouth starts closing after the unloading so that the velocity of the mandible is strongly limited.

There are several possibilities explaining this observation.

Firstly, *reflex events* possibly reduce the movement of the mandible after a sudden unloading. This idea is supported by Hannam et al. (1968) and Lamarre & Lund (1975) who show that after unloading of a static bite there is an inhibition of the jaw-closing muscles and an activation of the jaw-opening muscles.

Secondly, the rapid drop in force after unloading is possibly caused by *co-contraction* of the jaw-opening and -closing muscles during the bite. This hypothesis is put forward by Miles & Wilkinson (1982) who suggest that the major factor responsible for limiting the jaw closing movement is a weak contraction of the digastric muscles during the static phase before the unloading. When the resistance to the bite is suddenly withdrawn, the jaw closing movement is arrested by the so-called short-range stiffness (due to distortion of cross bridges between myofilaments; Rack and Westbury, 1974) in the co-contracting digastric muscles.

Thirdly, the *mechanical properties* of the jaw muscles, may also have profound effects on the dynamics of the bite force after the unloading. For example, if the sarcomeres of the jaw-closing and -opening muscles are below their optimal length at the start of the unloading, the *force-length properties* of the muscle fibers will cause a decrease in force of the jaw-closing muscles and an increase in force of the jaw opening-muscles during the jaw-closing movement. The *force-velocity properties* of the jaw muscles may also add to the reduction in force; the jaw-closing muscles lose a fair amount of their force when they shorten (less cross bridges are attached to actin filaments during the jaw movement, whereas, in general, the cross bridges also produce less force since their length reduces) and the jaw-opening muscles will gain force when they are stretched (cross bridges need to be broken to stretch the muscle and the stiffness in the muscle is quite high).

To get more insight into the relative contribution of the above mentioned phe-

nomena to the decrease in bite force after a sudden change in resistance we did a series of unloading experiments of the jaw and formulated a numerical model of the human jaw system simulating these unloading experiments.

In the unloading experiments subjects were asked to bite isometrically on two bars of which the resistance to the bite was suddenly withdrawn. Initial bite forces, initial mouth openings, distances of travel of the mandible when closing, as well as velocities of jaw closure were varied. To analyze possible effects of these variables on the force profiles after the unloading, the force exerted on the lower of the two bars and the position of the bar was measured during the experiments. We also recorded surface EMG's from the masseter muscle -that is one of the jaw-closing muscles-, and the digastric muscles -one of the jaw-opening muscles- to see how the level of (co-)contraction of these muscle influences the shape of the force profiles.

The numerical model of the human jaw system was used to judge the relative contribution the variables under discussion to the reduction in force, because the actual length and the velocity of the muscle fibers during the experiments are unknown.

Chapter 1 describes and discusses experiments attributed to the contribution of the (co-contracting) digastric muscles to the rapid decline in bite force magnitude after unloading of a static bite. This was done by asking subjects to perform two different biting tasks with sudden unloading, and correlating the degree of co-contraction of the digastric (as derived from their EMG's) with the impact force, the impact velocity (as measured after a travel distance of the mandible of 5 mm), and the residual force when the jaw system was in static conditions again after the impact.

Co-contraction of the digastrics was varied by asking subjects to perform the biting task while controlling bite force (force-controlled experiments) or jaw position (position-controlled experiments). In half of the experiments, subjects co-contracted their digastrics stronger in the position-controlled experiments as compared to the force-controlled experiments. However, there was no clear relationship between the level of co-contraction of the digastric muscles and the magnitude of the impact force, the impact velocity and the residual force. The results imply that co-contraction of the digastric muscles is not sufficient to explain the reduction in bite force and the low impact velocity after an unexpected jaw closing movement.

We also observed reflexes in the jaw-closing muscle and the jaw-opening muscles triggered by the onset of the unloading. Latencies of the reflex-events in the force-controlled conditions were comparable to those in the position-controlled conditions.

Chapter 2 reports on a study in which the magnitude of the impact velocity of the mandible -when the lower jaw is arrested after an unloading- is studied as function of the initial bite force, the initial degree of mouth opening and the distance of travel of the mandible. For that we calculated the velocity of the lower teeth at impact just before the mandible came to stand still in combinations of 4 different bite forces (100, 80, 60 and 40 N), 4 different initial degrees

of mouth opening (33.5, 30.5, 27.5 and 24.5 mm) and 3 different distances of travel of the mandible (4.5, 3.0 and 1.5 mm).

We found that the bite force rapidly declines after the unloading, resulting in a small impact velocity of the lower front teeth. This impact velocity largely depends on the magnitude of the initial bite force and the traveled distance; it is hardly sensitive to variations in degree of initial mouth opening. The maximal velocity of the lower teeth is 0.43 m/s (at an initial bite force of 100 N). It is calculated that if the bite force would not have declined, the mandible would have reached a velocity of 1.9 m/s (which is about 340 % higher than the velocity calculated from our experiments).

In chapter 3 a mathematical forward dynamic model of the human jaw system is described, that simulates static bites and the dynamic phase after unloading of this bite. With this model the influence of co-contraction, force-length properties and force-velocity properties of the jaw-opening and -closing muscles on the velocity at impact were explored. Morphometric data from a cadaver study were used in the model to come to anatomical realism. Physiological input values were mainly taken from literature. The remaining parameter values were found by fitting the model results optimally to the data from the different unloading experiments as descibed in chapter 2.

Model analyses show that the limitation of the jaw velocity is mainly due to the *force-velocity properties of the jaw-closing muscles*. These muscles dramatically lose their force when they shorten. This provides the jaw system with a quick mechanism for dealing with unexpected closing movements, in which neural control is always too late.

Moreover, it is shown that the force-length properties of the jaw muscles hardly contribute to the reduction in impact velocity. The compliance of tendinous sheets in the jaw muscles is unfavorable for the reduction in impact velocity, whereas co-contraction of jaw-opening and -closing muscles helps to limit impact velocity.

The second part of the thesis deals with events *when the jaw is arrested shortly after the unloading*. It is shown that after collision of the teeth, the remnant bite force - hereafter *residual force*- is remarkably small. For example, after an unloading of a 100 N bite, only 25% of the bite force is left when the mandible is arrested after a distance of travel of 4.5 mm. These low values of residual bite forces can not be explained by the force-velocity properties of the jaw muscles alone, since after cessation the lower jaw is in static conditions again.

Chapter 4 describes experiments dealing with the contribution of the initial bite force, the initial degree of mouth opening and the distance of jaw travel to the magnitude of the residual bite force. Furthermore, it is analyzed whether the low magnitude of the residual force can be attributed to reflex events of the jaw muscles or to the force-length properties of the jaw-closing muscles.

It is shown that the residual forces are largely dependent of the distance of jaw travel (large travel distances give small residual forces) and are barely sensitive to variations in initial mouth opening. Rela-

tively, the value of the residual force is independent of the magnitude of the initial bite force. For example: a bite force of 100 N as well as a bite force of 40 N are both reduced to 60 % of their initial value after the jaw is arrested after a distance of jaw travel of 1.5 mm, and to 25 % of their initial value after a 4.5 mm jaw travel.

The low values of the residual forces can *not* be attributed to reflex events, since the sum of reflex latencies and electro-mechanical delay of the jaw muscles puts a substantial loss in muscle force well after the maximum in the residual force was measured. [It takes about 80 ms for the masseters to decrease their force to a 50% level when their excitation is switched off, and the masseter muscle shows silent periods with latencies of at least 9 ms, whereas the maximum of the residual forces is at about 35 ms after the unloading.]

Finally, in chapter 4 it is shown that the force-length properties of the jaw-closing muscles are *not* responsible for the small values of the residual forces, since over the trajectories used in the descibed experiments, the sarcomere lengths of the jaw-closing muscles are *beyond* their optimum sarcomere length.

The low values of the residual forces may partly be explained by the force-velocity properties of the jaw-closing muscles. After the jaw is arrested, the muscle fibers of the jaw-closing muscles may still shorten for a while (due to the in series arrangement of the muscle fibers and the tendinous sheets and to differences in their mechanical properties). As long as these internal movements occur (tendon creep) this will give loss in force (particularly immediately after the jaw movement).

For that we investigated in chapter 5 the influence of the velocity of jaw closure on the force profile of the closing mandible after an unloading. Data are presented on unloading experiments of a 100 N static bite at an initial mouth opening of 23.5 mm over four different distances of jaw closure. These experiments were performed twice, with and without the application of a damping resistance to jaw closure. The maximal velocity of jaw closure was reduced by a factor of five in the slow jaw closing movement in comparison with the fast ones.

It is shown that after unloading the decline in bite force during jaw movement is largely dependent on the velocity of jaw closure. This is in good agreement with our hypothesis that the force-velocity properties of the jaw-closing muscles are mainly responsible for the decline in bite force during jaw closure. The magnitude of the residual force is insensitive to variations in closure velocity. This suggests that the effects of the force-velocity properties in conjunction with tendon creep are *not* long-lasting enough to account for the small residual forces.

It is suggested that the low value of the residual forces is due to *history-of-shortening-dependent changes in the active contractile system*. We think that the low residual forces are brought about by either (1) a non-uniform sarcomere behavior of the jaw-closing muscles when contracting or (2) a long lasting change in the myofilament system of the closing muscles induced by the sudden shortening of muscle fibers.

Samenvatting

In dit proefschrift wordt de fysiologie behandeld die ten grondslag ligt aan de snelle afneming in bijtkracht wanneer de weerstand tegen een krachtige beet plotseling wordt weggenomen.

Dit is een belangrijk onderwerp aangezien bijtkrachten groot kunnen zijn (in de literatuur worden bijtkrachten van 600 N vermeld) en de effectieve massa van de onderkaak slechts ca. 0.20 kg is (ter plaatse van de ondertanden), zodat er het risico bestaat dat de tanden beschadigd worden wanneer men door hard, bros voedsel bijt. In theorie bestaat de mogelijkheid dat, wanneer men met een constante kracht van 100 N een noot van 2 cm doorsnede kraakt, de kaken op elkaar slaan met een snelheid van 4.5 m/s (dat is 16 km/uur) wanneer de noot breekt.

In werkelijkheid gebeurd dit niet. Dit heeft men laten zien met proeven waarbij experimenteel de weerstand tegen een krachtige beet plotseling werd weggenomen. Uit deze zogenaamde "unloading" proeven (hierna: "ontlastingsproeven") blijkt dat na het vrijgeven van de beet de bijtkracht plotseling zeer snel afneemt. Hierdoor is de snelheid van de onderkaak laag wanneer de tanden opelkaar slaan.

Er zijn verschillende verklaringen voor deze snelle afneming in bijtkracht.

Ten eerste is het mogelijk dat *reflexen* zorgen voor de snelle afneming in bijtkracht na ontlasting van de beet. Dit idee wordt ondersteund door de waarnemingen van Hannam et al. (1968) en Lamarre & Lund (1975). Deze auteurs laten zien dat de activiteit van de kaaksluitspieren afneemt, en die van de kaakopeningsspieren toeneemt wanneer de beweging van de onderkaak begint.

Ten tweede kan de snelle afneming in bijtkracht na ontlasting van de beet worden veroorzaakt door een *co-contractie* van sluit- en openingsspieren gedurende de beet. Dit idee werd in 1982 geopperd door Miles and Wilkinson. Zij suggereren dat een zwakke contractie van de openingspieren gedurende de statische fase van de beet de beweging van de onderkaak afremt wanneer deze plotseling wordt vrijgegeven. Het verschijnsel zou gebaseerd zijn op verkleving van de verbindingsbruggen tussen de contractiele elementen van de actieve spiervezels van de openingspieren ("short-range stiffness", Rack and Westbury, 1974).

Ten derde kunnen ook de *mechanische eigenschappen* van de kaakspieren zorgen voor de snelle afneming in bijtkracht na ontlasting van de beet. De *kracht-lengte-eigenschappen* van de sluit- en openingspieren kunnen voor krachtafneming zorgen wanneer de lengte van de sarcomeren van deze spieren kleiner is dan hun optimale lengte wanneer de onderkaak wordt ontlast. De kracht van de sluitspieren neemt dan af, en die van de openingspieren neemt toe wanneer de kaak in beweging komt. Mogelijk zijn de *kracht-snelheid-eigenschappen* van de spiervezels ook van belang voor het wegvallen van de bijtkracht. Bij het sluiten van de kaak kunnen de sluitspieren veel kracht verliezen (tijdens de beweging kunnen minder verbindingen tussen de contractiele elementen ontstaan en de gemiddelde lengte van de verbindingen neemt af); de openingsspieren kunnen extra kracht ontwikkelen wanneer ze gerekt worden (de gemiddelde lengte van de verbindingen neemt toe).

Tot beter begrip van de relatieve bijdrage van bovengenoemde factoren aan de afneming in bijtkracht -na een plotselinge ver-

andering van de bijtweerstand tijdens krachtig bijten- zijn een aantal ontlastingsproeven van de onderkaak gedaan. Daarnaast is een numeriek model van het menselijke kauwsysteem geformuleerd, waarmee de experimenten kunnen worden gesimuleerd.

De ontlastingsproeven werden gedaan met behulp van een apparaat dat bestaat uit twee staven, waarvan de onderste kan scharnieren en de bovenste vast is. Door verschuiving van de bovenste staaf kon de afstand tussen de staven worden gevarieerd. De kracht uitgeoefend op de onderste staaf en de positie van deze staaf kon worden gemeten. Proefpersonen werd gevraagd op de staven te bijten waarna, bij een zekere grootte van bijtkracht, de onderste staaf plotseling losschoot en naar boven scharnierde. De onderkaak kwam hierdoor plotseling vrij en sloot zich. Er werden series van proeven gedaan waarbij de initiële bijtkracht, de initiële mondopening, de grootte van de kaakbeweging na het wegvallen van de belasting van de onderkaak, en de snelheid van mondsluiting werd gevarieerd.

Soms werden tijdens de proeven electromyogrammen (EMG's) van de m. masseter (dat is één van de sluitspieren) en van de m. digastricus anterior (dat is een van de openingspieren) gemaakt om te kunnen zien hoe de mate van (co)-contractie van deze spieren de gemeten krachtsprofielen beïnvloedde.

Het numerieke model van het menselijke kauwsysteem werd gebruikt om de relatieve bijdrage van bovengenoemde variabelen aan de afneming in bijtkracht te bestuderen, aangezien de lengte en snelheid van de contracterende spiervezels tijdens te proeven onbekend is, en moeilijk is te meten.

Hoofdstuk 1 beschrijft proeven die zich richten op de effecten van de mate van co-contractie van de mm. digastrici op de afneming in bijtkracht na ontlasting van een statische beet. Proefpersonen werd gevraagd twee verschillende bijttaken uit te voeren met een plotseling wegvallen van de bijtweerstand; de activiteit van de mm. digastrici werd geregistreerd en gecorreleerd met de inslagkracht en inslagsnelheid van de onderkaak (vlak voor dat deze na een sluitbeweging van 5 mm werd gestuit), en met de restkracht die gemeten werd zo'n 35 ms nadat de onderkaak weer tot rust gekomen was. De (co)-contractie van de mm. digastrici werd gevarieerd door de proefpersonen te vragen (1) tijdens de proeven de bijtkracht monotoon te vergrootten (force-controlled experiments) of (2) hun openingsspieren bewust aan te spannen tijdens de beet en hun mond open te houden wanneer de weerstand tegen de beet wegviel (position-controlled experiments). In ongeveer de helft van de proeven contraheerden de proefpersonen hun digastrici sterker in de positiegestuurde proeven; sterkere co-contractie van de openingsspieren bleek echter niet samen te gaan met een kleinere inslagkracht, een kleinere inslagsnelheid en een kleinere restkracht. Dit betekent dat co-contractie van de openingsspieren nauwelijks bijdraagt aan de afneming in bijtkracht na het wegvallen van de bijtweerstand.

We vonden ook reflexactiviteit in electromyogrammen van de m. masseter en de m. digastricus als antwoord op het wegvallen van de bijtweerstand. We vonden geen duidelijke verschillen in latentietijden, reflexgrootte en -duur tussen de twee verschillende bijttaken.

Hoofdstuk 2 beschrijft proeven die inzicht geven in de relatie tussen de grootte van de inslagkracht van de onderkaak, en de initiële bijtkracht, de initiële mondopening en de grootte van de kaakbeweging na het wegvallen van de weerstand van de onderkaak. Inslagkrachten werden gemeten en kaaksnelheden berekend voor alle combinaties van 4 verschillende initiële bijtkrachten (100, 80, 60 and 40 N), 4 initiële mondopeningen (33.5, 30.5, 27.5 and 24.5 mm) and 3 verschillende bewegingsafstanden van de onderkaak (4.5, 3.0 and 1.5 mm).

De snelheid van de onderkaak op het moment van inslag bleek sterk afhankelijk te zijn van de grootte van de initiële bijtkracht en de grootte van de afgelegde bewegingsafstand van de onderkaak; de inslagsnelheid van de onderkaak bleek nauwelijks beïnvloed te worden door variatie in mondopening. Na ontlasting van een 100 N beet bereikt de onderkaak na 12 ms zijn maximale snelheid (0.43 m/s); de onderkaak heeft dan een traject van 4 mm afgelegd. Ter vergelijking: onder gelijke omstandigheden maar met een bijtkracht die niet afneemt zou de inslagsnelheid van de onderkaak 1.9 m/s zijn (dit is 340% hoger dan de snelheid berekend uit de experimentele gegevens).

In hoofdstuk 3 wordt een mathematisch model van het humane kauwstelsel beschreven. Dit model simuleert bijtkrachten na het wegvallen van de bijtweerstand tot het moment dat de onderkaak wordt gestuit. Met dit model werd de invloed van reflexen, co-contractie, en de kracht-lengte- en kracht-snelheid-eigenschappen van de sluit- en openingsspieren op de inslagsnelheid van de kaak bestudeerd. Om het model zo realistisch mogelijk te maken werden morfometrische data (verkregen door middel van metingen aan menselijke kadavers) als anatomische input gebruikt. Fysiologische data werden uit de literatuur genomen, terwijl de overige parameter gevonden werden door het model te optimaliseren aan de hand van de resultaten van de serie experimenten, zoals beschreven in hoofdstuk 2.

Analyses met het model tonen aan dat voornamelijk de *kracht-snelheid-eigenschappen van de sluitspieren* verantwoordelijk zijn voor de snelle afneming in bijtkracht en dus voor de lage snelheden van de onderkaak op het moment van inslag. De sluitspieren verliezen bijna al hun kracht wanneer zij verkorten. Deze eigenschap zorgt er voor dat de onderkaak automatisch en snel -dat wil zeggen zonder invloed van het centrale zenuwstelsel- bijtkracht verliest wanneer de onderkaak bij krachtig bijten plotseling doorschiet.

Met behulp van het model konden we ook aantonen dat de kracht-lengte-eigenschappen van de kauwspieren nauwelijks de inslagsnelheid van de onderkaak kunnen beïnvloeden. Het blijkt dat de compliantie van de peesplaten in de kauwspieren ongunstig is voor de inslagsnelheid (hoe hoger de compliantie, hoe groter de snelheid). Meer co-contractie van sluit- en openingsspieren is gunstig: dit veroorzaakt een kleinere inslagsnelheid van de onderkaak.

Het tweede deel van het proefschrift behandeld de bijtkrachten die gezien worden *na afloop van de dynamische fase*. Zoals reeds opgemerkt zijn deze *restkrachten*, wanneer de onderkaak niet meer beweegt, opmerkelijk klein. Bijvoorbeeld, van een initiële bijtkracht van 100 N is slechts 25% over nadat de onderkaak na een bewegings-

afstand van 4.5 mm is gestopt. Dit verschijnsel kan niet alleen verklaard worden uit de kracht-snelheid-eigenschappen van de kaakspieren (immers, de onderkaak beweegt niet meer).

Hoofdstuk 4 beschrijft proeven die antwoord geven op de vraag hoe de grootte van de initiële bijtkracht, de initiële mondopening, en de bewegingsafstand van de onderkaak, de grootte van de restkracht beïnvloeden. Bovendien werd onderzocht of reflexen de waarde van de restkracht beïnvloeden, en wat de bijdrage is van de kracht-lengte-eigenschappen van de sluitspieren aan de grootte van de restkracht.

Onze proeven laten zien dat de grootte van de restkracht voornamelijk afhankelijk is van de grootte van de sluitbeweging (hoe groter de bewegingsafstand, hoe kleiner de restkracht), terwijl de mate van mondopening bij het begin van het experiment nauwelijks invloed heeft op de grootte van de restkracht. De relatieve grootte van de restkracht blijkt onafhankelijk te zijn van de grootte van de initiële bijtkracht. Zowel bij een initiële bijtkracht van 100 N als van 40 N is de grootte van de restkracht 60% van de initiële bijtkracht wanneer de kaak wordt gestopt na een bewegingsafstand van 1.5 mm, en 25% wanneer de onderkaak na 4.5 mm wordt tegengehouden.

De lage waarden van de restkrachten kunnen *niet* toegeschreven worden aan reflexactiviteit. Door de latentietijden van reflexen en de electro-mechanische vertraging van de kauwspieren zijn de effecten van reflexactiviteit veel te laat om de restkracht duidelijk te kunnen beïnvloeden. [Bij 100 N experimenten valt de electrische activiteit van de m. masseter stil na een latentietijd van *ca.* 10 ms. Daarna duurt het *ca* 80 ms voordat de kracht van de m. masseter tot 50% gedaald is (de electro-mechanische vertraging). De restkracht heeft 35-40 ms na de start van de kaakbeweging zijn hoogste waarde bereikt.]

In hoofdstuk 4 wordt eveneens aangetoond dat de kracht-lengte-eigenschappen van de sluitspieren *niet* verantwoordelijk zijn voor de lage waarde van de restkracht, aangezien de lengte van de sarcomeren van de sluitspieren groter is dan de optimale lengte bij de start van de kaakbeweging.

De kracht-snelheid-eigenschappen van de kauwspieren kunnen de lage waarden van de restkrachten (voor een gedeelte) veroorzaken. Aan deze hypothese ligt de idee ten grondslag dat, wanneer de onderkaak wordt gestuit, de spiervezels niet direkt tot rust komen maar nog een tijdje doorgaan met samentrekken (ten gevolge van de in-serie-ordening van spiervezels en peesplaten, en de verschillen in mechanische eigenschappen tussen spiervezels en peesplaten). Zolang er in de spier nog beweging plaatsvindt tussen de spiervezels en de peesplaat ("kruip van de peesplaat") is er minder spierkracht. Dit verschijnsel is waarschijnlijk het sterkst direkt nadat de onderkaak tot stilstand is gebracht. Om dit idee te falsificeren hebben wij proeven gedaan die een mogelijke relatie tonen tussen de snelheid van kaaksluiting (en dus de kracht-snelheid-eigenschappen van de kauwspieren) en de grootte van de restkracht.

Deze proeven zijn beschreven in hoofdstuk 5. In dit hoofdstuk worden ontlastingsproeven van een initiële beet van 100 N bij een initiële mondopening van 23.5 mm beschreven bij vier verschillende bewe-

gingsafstanden van de kaak. De proeven werden twee maal uitgevoerd, waarbij de snelheid van de mondsluiting wordt gevarieerd. Door de beweging van de onderste bijtstaaf met behulp van een hydraulische demper te vertragen in de dempingexperimenten werd de maximale snelheid van kaaksluiting met een factor vijf verminderd.

Het bleek dat de afneming in bijtkracht na de ontlasting van de beet sterk afhankelijk is van de snelheid van kaaksluiting; dit ondersteunt onze hypothese dat de kracht-snelheid-eigenschappen van de sluit-spieren voornamelijk de dynamiek van de bijtkracht in de dynamische fase na ontlasting van de beet bepalen. De grootte van de restkracht, nadat de onderkaak tot stilstand is gekomen, bleek ongevoelig te zijn voor de variatie in snelheid van mondsluiting; kennelijk kunnen de kracht-snelheid-eigenschappen van de spieren samen met kruip van de peesplaat de lage waarde van de restkracht na afloop van de bijtproef *niet* verklaren.

Wij veronderstellen dat aan de lage waarden van de restkracht verschijnselen ten grondslag liggen die gevoelig zijn voor de "*hoeveelheid verkorting die heeft plaatsgevonden" in de actieve contractiele elementen* van de sluitspieren; mogelijk worden de kleine waarden van de restkrachten veroorzaakt door (1) inhomogeniteit in de sarcomeren van de sluitspieren tijdens contractie of door (2) langdurende veranderingen in de myofilament systemen van de sluitspieren ten gevolge van een plotselinge verkorting van de spiervezels.

Curriculum vitae

List of publications

Dankwoord

Curriculum Vitae

Na het basisonderwijs begon de auteur in 1977 met een rustige start op de MAVO, 4 km van haar geboorteplaats Uithuizen. Na de MAVO sprong ze via 1 jaar HAVO naar Atheneum 5 en 6. Nadat ze in 1984 uitgeloot was voor de studie Geneeskunde, en Wiskunde haar toch wel erg taai leek, begon ze op de Academie voor Fysiotherapie. Na afloop van haar studie werkte zij gedurende 1 jaar fulltime als fysiotherapeut in diverse particuliere praktijken. Daarna studeerde zij Bewegingswetenschappen in Groningen van 1989-1992. Gedurende haar studie was ze met name geïnteresseerd in biomechanica en wiskundige modelvorming. Haar afstudeerproject was het vertalen van EMG-signalen in kracht door middel van een computermodel.

Na afloop van haar studie werkte ze samen met Bert Otten gedurende 1 jaar aan een computer simulatie van de schaatsbeweging. In november 1993 begon ze met haar promotieonderzoek, welke nu is afgerond in een dissertatie.

Gezien het onderzoek slechts voor 2 jaar werd gefinancierd verrichtte ze veel bijbaantjes. Ze geeft sinds 1992 de cursus 'blessure preventie in de sport' op de Academie voor Lichamelijke Opvoeding. Daarnaast heeft ze op deze opleiding gedurende 1 jaar biomechanica en kinesiologie gegeven. Zomers is ze werkzaam als fysiotherapeut.

Sinds 1 augustus 1997 is ze werkzaam als onderwijscoördinator en docent bij de vakgroep Anatomie in Groningen. Daar geeft zij onderwijs in anatomie en kinesiologie aan studenten Bewegingswetenschappen en Geneeskunde.

Articles (international journals)

Nagashima T, Slager GEC, Otten E, Broekhuijsen ML and van Willigen JD (1997). Impact velocities of the teeth after a sudden unloading at various initial bite forces, degrees of mouth opening and distances of travel. *Journal of Dental Reseach, 76, 1751-1759.*

Slager GEC , Otten E, van Eijden TMGJ and van Willigen JD (1997). Mathematical model of the human jaw system simulating static biting and movements after unloading. *Journal of Neurophysiology 78, 3222-3233, 1997.*

Slager GEC , Otten E, Nagashima T and van Willigen JD (1998). The riddle of the large loss in bite force after fast jaw-closing movements. *Journal of Dental Reseach (accepted).*

Slager GEC , Otten E, Nagashima T and van Willigen JD. Human bite force responses after fast and slow jaw closing movements. *Journal of Dental Reseach (submitted).*

Willigen JD van, Otten E, Slager GEC and Broekhuijsen ML (1997). Contribution of the digastric muscles to the control of bite force in man. *Archives of oral Biology, 42, 45-56.*

Articles (Dutch journals)

Mulder P, Smit AJ, van Slochteren-van der Boor JC, Kuipers WD en Slager G (1989). Het effekt van 2 Hz T.E.N.S. op de perifere circulatie bij patienten met M. Raynaud. *Nederlands Tijdschrift voor Fysiotherapie, 99, 326-331.*

Books

Slager GEC (1994). Computermodellen binnen bewegingswetenschappelijk onderzoek? In: '*Terugblikken en vooruitzien*', symposium 10-jarig bestaan werkgroep Bewegingswetenschappen Groningen. JG Bos, HME Hendrickx, K Reynders and GA Schäperclaus, Groningen, pp. 153-159.

Slager GEC, Otten E, Van Willigen JD and Broekhuijsen ML (1995). Contribution of mechanical properties of jaw muscles to the control of unexpected jaw-closing movements. In: *Proceedings of the Osaka International Oral Physiology Symposium on Brain and Oral Function.* T Morimoto, T Matsuya and KJ Takada eds., Excepta Medica International Congress Series 1079, pp. 369-373.

Willigen JD van, Slager GEC, Broekhuijsen ML and Otten E (1995). Digastric muscle responses as a function of knowledge of the task to be performed. In: *Proceedings of the*

Osaka International Oral Physiology Symposium on Brain and Oral Function. T Morimoto, T Matsuya and KJ Takada eds., Excepta Medica International Congress Series 1079, pp. 205-216.

Abstracts

Otten E and Slager GEC (1994). A mathematical model of speedskating. Symposium of the Society of Experimental Biology. Swansea. *(abstract book)*

Slager GEC and Otten E (1993). Efficient speed skating, -presentation of a computer model. Proceedings of the Young Physiologist Meeting, *Eur.J. of Physiol., Pflügers Arch,* 424: R1-R10.

Slager GEC and Otten E (1993). Efficient speed skating. Congress: *Basic and applied exercise physiology*, University of Nijmegen, the Netherlands, 10-11 june 1993, p 161.

Slager GEC, Otten E, Eijden TMGJ van, Willigen JD van and Nagashima T (1997). Not stiffness control, but muscle properties prevent high dental impact velocities after unexpected jaw-closing movements. Store Kro, the Netherlands. *J. Oral Rehabilitation (submitted).*

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